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The Massachusetts Medical Society

THE MECHANISM OF EMOTIONAL DISTURBANCE OF BODILY FUNCTIONS*

BY W. B. CANNON, M.D.†

IN 1896, when I was a first-year medical student, Professor Henry P. Bowditch, whose memory his former students delight to recall, invited me to make use of the then newly discovered Röentgen rays in a study of the activities of the alimentary canal. In December of that year we demonstrated to the members of the American Physiological Society the passage down the esophagus of a swallowed mass made opaque to the rays by adding subnitrate of bismuth. After that beginning we studied the mechanical functions of the stomach and intestines, and the various conditions affecting the rate of passage of food through the digestive tract. Almost from the start of these investigations an outstanding fact appeared. The smooth-running recurrent waves of peristalsis coursing over the stomach, and the rapidly shifting segmentation of the food masses in the small intestine were promptly abolished whenever the subject showed signs of anxiety, distress or rage. It was evident that these alimentary functions were extremely sensitive to emotional disturbances. My interest in effects of excitement, which was thus initiated, led to studies of the services of the sympathetic nervous system, by itself and in coöperation with glands of internal secretion, and that in turn to an examination of the parts of the central nervous system which govern these fundamental reactions of the organism. You will pardon these references to personal experience, I trust, for they account for the selection of the title of this discourse. Since you have asked a physiologist to address you, you will permit him, I feel sure, to come and bring his contribution to medical thought and counsel. It has seemed to me, therefore, that we might profitably consider together the ways in which strong emotional states may endanger bodily welfare.

I think that we must admit that, although physicians have not infrequent occasions to observe instances of functional disturbance due to emotional excitement, there is an inclination to minimize or to slight that influence, or even to deny that it is part of a physician's service to his patient to concern himself with such troubles. Let the patient go to the clergyman for comfort and consolation and for the resolution of his deep anxieties. A too common unwillingness among physicians to regard seriously the emotional elements in disease seems to me to be due perhaps to the subtle influence of two extreme attitudes and disciplines. On the one hand is the powerful impress of morphological pathology. So triumphantly and so generally has it demonstrated under the microscope the structural alterations which accompany altered functions that any state which has no distinct "pathology" appears to be unreal or of minor significance. Fears, worries and states of rage and resentment leave no clear traces in the brain. What, then, have we physicians to do with them? On the other hand, these mysterious and dominant feelings which surge up within us from unknown sources—are they not pure perturbations of the "psyche"? In that case, what, again, have we physicians to do with them? If we show this indifference, however, is it surprising that men and women, beset by emotional stresses, turn from us and go for help to faith healers, to Christian Scientists and to others who recognize the reality of these disturbing states?

An escape from the insistent demands of the pathologist for morphological evidence of disease, and also from the vagueness and mysticism of the psychological healers, can be found, I am convinced, in an understanding of the physiological processes which accompany profound emotional experience. As a physiologist I have the reasonable right to consider what goes on in the nerve paths of the brain as not associated with any demonstrable structural change. Indeed, very pronounced and disastrous consequences may result in the organism because of habit reactions, which may be regarded as not different in quality from any of our ordinary ways of behaving. Also as a physiologist I have the reasonable right to regard suddenly altered functions of organs innervated from the central nervous system as occurring in consequence of nerve impulses discharged from that system.

*The Annual Discourse delivered before the Massachusetts Medical Society, at Worcester, June 6, 1928.

†For record and address of author see "This Week's Issue," page 920.

Using the physiological point of view, therefore, I propose to consider emotions in terms of nerve impulses, much as I might consider the nerve impulses from the "motor area" of the cerebral cortex as they govern the movements of skeletal muscles. Although I shall use words with psychological implications, such as "fear," "rage," "feelings," and others, let me state at the outset that I use them solely as convenient short terms for complex activities in the brain. I shall be discussing, throughout, the *physiological* aspects of emotional excitement—the nervous mechanisms which are operating.

First, what is an emotion? From the physiological point of view it is a typical reaction pattern. Let us consider rage as an example. In its extreme form the signs of rage include the crouching body, the moist or frowning brow, the firm lips, the clenched or grinding teeth, the growled threats or imprecations, and the tightened fists or the seized weapon ready for attack. This is a complex attitude which we do not have to learn—its occurrence is a part of our native inheritance. It occurs promptly when the stimulus is appropriate. It is a constant and uniform type of behavior, having features which are common in widely scattered races of men and even in lower animals, so that the nature of the attitude is at once understood without the necessity of words. It is a permanent mode of reaction; throughout an individual's life the characteristic display of the rage response may be suddenly evoked in all its elaborateness and, whether in childhood or old age, it differs only in minor details. Further, it is a response to a fairly definite stimulus—any hampering or checking of activity, or opposition to one or another primary impulse brings it out. Threaten the free motion of a dog or a man and the teeth will be uncovered. Again, the rage response may be interpreted as being useful. Elsewhere[1] I have called attention to the wide range of bodily adjustments which occur when one is enraged—the more rapid heart-beat, the redistribution of the blood, the increase of red blood corpuscles in the circulation, the larger ventilation of the lungs, the dilatation of the bronchioles, the liberation of sugar from the liver, the secretion of adrenin with its favorable action on fatigued muscles—all of which may properly be regarded as rendering the organism more efficient in struggle, in such struggle as may be required to overwhelm the opposition and to allow the natural impulse to prevail. As we survey the characteristics of the outburst of rage as a typical emotion—the inborn, prompt, constant, uniform, permanent and useful nature of the response to a definite kind of stimulus—we note that these are the characteristics of a simple reflex, such as sneezing or coughing. They differ not in quality but in complexity.

Man is superior to the lower animals mainly because of the extensive development of the cerebral hemispheres. Comparative anatomy shows that these structures have been superposed on a brain stem which differs relatively little in the higher vertebrates. And physiological investigation has proved that whereas the reactions which involve the cerebral cortex may be delayed, unpredictable, short-lived, and readily modifiable, those which involve the lower levels of the brain and spinal cord are prompt, uniform and stereotyped. Hence the difference between the complex behavior of the normal human being and the relatively simple behavior of the idiot. It is of interest, therefore, to learn where the nervous mechanisms lie which operate the various emotional displays. Do these mechanisms have their seat in the newly developed cerebral cortex or in the more ancient parts of the brain?

In the brain stem are centers which, in the lower vertebrates, lacking a cerebral cortex, carry on the primitive functions of maintaining existence, such as seizing their prey and escaping from their enemies. These are activities which in man are associated with attack or with flight from danger and are attended by the emotions of rage or fear. In higher forms the centers for these functions, though normally held in check by the dominant cortex, are capable of energetic response when conditions require urgent and insistent action. It seemed reasonable to expect that the centers in the brain-stem would manifest their typical activity if the cerebral cortex was removed. Removal of the cortex would destroy the possibility of sensation and, therefore, a depressing or disturbing anesthetic could be dispensed with. Accordingly Britton and I[2], using cats as subjects, undertook an investigation of some of the immediate effects of a decortication which left intact almost all of the gray masses at the base of the brain. As soon as recovery from anesthesia was complete a remarkable group of activities appeared, such as are usually seen in an enfuriated animal—a sort of sham rage. These quasi-emotional phenomena, which appeared to result from the restraint, included lashing of the tail, arching of the trunk, thrusting and jerking of the restrained limbs, display of the claws and clawing motions, snarling and attempts to bite. These were all actions due to skeletal muscles. Besides these, and more typical and more permanent, were effects on the viscera, produced by impulses discharged over the sympathetic nerve fibres. They included erection of the tail hairs, sweating of the toe pads, dilatation of the pupils, micturition, a high blood pressure, a very rapid heart beat, an abundant outpouring of adrenin, and an increase of blood sugar up to five times the normal concentration[3]. This display of a "pseudaffective" state or sham rage might continue for two or three hours.

As stated above, Britton and I left untouched almost all of the basal gray matter of the anterior brain-stem. Where among these basal ganglia does the neurone pattern for the rage

response reside? The answer to this question was obtained by Bard[4] who, after removing under ether the cerebral cortex and various amounts of the brain-stem, studied the behavior of the preparation. He found that typical sham rage, accompanied by vigorous discharge of sympathetic impulses, occurs when both hemispheres, the corpora striata and the anterior half of the diencephalon have been completely isolated (i. e., the crosshatched parts in figure 1).

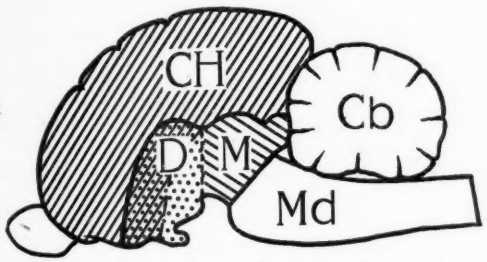

FIGURE 1. Median section of the brain. CH, cerebral hemispheres; D, diencephalon (indicated by dots); M, mesencephalon; Cb, cerebellum; Md, medulla. The crosshatching, from right downward to left, marks the portion of the brain which can be removed without interfering with the emotional expression of rage.

The additional extirpation of the posterior half of the diencephalon promptly abolishes the spontaneous activity. Further tests proved that the center lies in a small brain mass in the ventral part of this region, i.e., in the subthalamus.

Here is a fundamental fact which I wish to emphasize—that the nervous organization for the display of rage, both in bodily attitudes and in visceral changes, is located in an ancient portion of the brain, the optic thalamus which is a part of the diencephalon. This region is not like the cerebral cortex where new adjustments with the outer world are constantly being made or modified. Instead, it is like the spinal cord, a place where the simpler mechanism for orderly motions reside and where stimulation evokes fixed and uniform reflex responses. The typical postures and visceral changes which result from action of the thalamus are more complicated than the knee jerk or other spinal reflexes, but they are not essentially different.

I have laid stress on the locus of the physiological mechanism for the reflex figure of rage because it may serve as a model for other primitive emotional responses. The expressions of fear, joy and grief are similar to it in character. In their essential features they are not learned (i. e., they are inborn) and they are prompt, constant, uniform and permanently established patterns of reaction to appropriate stimuli. In other words they are like the simple reflexes and not like the complicated adjustments managed by the cortex. There is good evidence that the central control for the expression of these emotions, like that for rage, lies in the thalamic region. For example, Bechterev[5] has reported that in an animal freshly deprived of its cerebral hemispheres, petting may call forth signs of pleasure, e. g., purring in the cat and tail wagging in the dog.

The evidence which I have adduced to show that the neural arrangement for emotional display is near the optic thalamus has been based wholly on experiments on lower animals. That evidence, however, is consistent with indications that in man also emotional expression is managed by parts of the brain below the cortex and specifically by centers in or near the optic thalamus. Thus when in human beings the cortical processes are abolished by anesthesia, emotional display may be most remarkable. During the excitement stage of anesthesia, for example, the patient may sob as in grief, or laugh as in joy, or make the energetic aggressive actions of rage. While the patient is struggling, shouting and muttering the surgeon may open the chest or perform other operations of equal gravity; a few minutes later, when conscious, the patient will testify that he has been wholly unaware of what has happened. It is when "laughing gas" or alcohol has set aside the cortical functions (i. e., has functionally decorticated the individual), that he laughs or weeps. In all these conditions the drug acts first as a depressant on the highly sensitive cells of the cortex and thus lessens or temporarily destroys their control of lower centers; then the lower centers, released from the dominance of the cortex as in surgically decorticated animals, show forth their functions in free play.

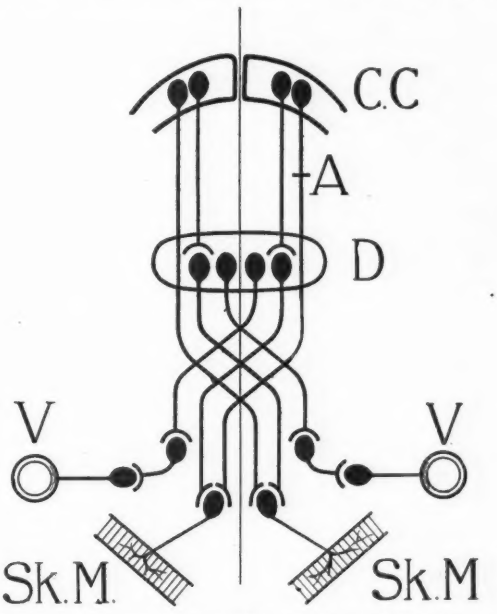

FIGURE 2. Diagram of possible relations of the nerve cells of the cerebral cortex (C.C.) and of the thalamic portion of the diencephalon (D.) to the viscera (V.) and to skeletal muscles (Sk.M.). The cortico-thalamic fibre is regarded as inhibitory. Sensory fibres are not represented. Damage to the cortico-spinal tract at A interrupts cortical control of certain skeletal muscles on one side, but it does not prevent control of these muscles on both sides by the centers in the diencephalon. Unilateral injury of centers in the diencephalon may leave bilateral control from the cortex.

In harmony with the experimental evidence from lower animals and the just described pharmacological evidence from man is that derived from pathological studies of human cases. In certain forms of hemiplegia patients are in-

capable of moving the face on the paralyzed side; but if an emotional (i. e., a sorrowful or joyous) situation develops, the muscles which were unresponsive to voluntary (i. e., cortical) control flash into action and give *both* sides of the face the expression of sadness or gaiety[6]. These are cases of subcortical interruption of the motor tract (e. g., at A. figure 2), and presence of an intact optic thalamus. The converse of this condition is seen in unilateral injury of the thalamic neurones (figure 2); then the patient moves symmetrically both sides of the face at will, but when he laughs or weeps the emotional expression is unilateral. Cases of pseudo-bulbar palsy also bring interesting testimony. In this disease there is usually a bilateral facial paralysis, with one side somewhat more involved than the other. Voluntary pursing of the lips as in whistling, or wrinkling of the forehead, or making a grimace, may be impossible. And yet the apparently paralyzed muscles function quite normally in laughing or crying, scowling or frowning. Indeed patients may have prolonged and uncontrollable fits of laughing or weeping. According to Brissaud[7] the pathological condition in this disease is a lesion of a part of the cortico-thalamic tract which frees a portion of the thalamus from the cortical check. All these observations, experimental and clinical, consistently point to the optic thalamus as the region in which resides the neural organization for the different emotional expressions.

The thalamic region is not only the seat of the neural patterns for the various emotional displays. It appears to be also the source of the peculiar feelings which contribute glow and color to otherwise drab sensations. The evidence for this inference is mainly clinical. Head[8] has cited numerous cases of unilateral lesions in the thalamic region in which stimuli which evoke feelings have an excessive effect—pin pricks, painful pressure, pronounced heat or cold all produce much more distress on the damaged side than on the normal side of the body. Agreeable stimuli likewise are felt keenly on the damaged side; a warm test tube, for example, may give rise to intense pleasure, attended by signs of enjoyment on the face and by exclamations of delight. Again, the playing of music and the singing of hymns may arouse such increased emotional feeling, which is referred by the patient to the damaged side, that they may be intolerable. Imagined or remembered situations associated with past emotional experiences have an influence on the damaged side similar to the disturbing stimuli from the sense organs. This excessive influence of affective stimuli, whether from the body surface or from the cortex, Head attributed to the release of the thalamus from cortical control. When freed from check it overacts. And since in these cases the feelings are magnified on the damaged side, Head has concluded that the thalamus is occupied with the emotional aspect of sensation and that the unilateral overaction there is the cause of the unilateral magnification of feeling.

We have reviewed the evidence that the neurones of the thalamic region discharge outward and downward to muscles and viscera to produce the typical bodily changes of emotional excitement, and that they discharge upward to the cortex to add richness and warmth to the simple sensations. Two other important points I wish now to emphasize.

The first of these is concerned with the relations of the cortical and the thalamic control of bodily processes. It is clear that *skeletal muscles are governed at both levels, cortical and thalamic* (see figure 2); for example, we may laugh spontaneously because of a ludicrous situation (thalamic laughter) or we may laugh as a voluntary act (cortical laughter). It is quite as clear that the *viscera*, on the other hand, *are only under thalamic government;* we cannot by direct act of will increase the blood sugar, accelerate the heart, or stop digestion. When there is double control the cortical neurones, to be sure, are ordinarily dominant and may not release the excited neurones of the thalamus (though we sometimes cry or laugh "in spite of ourselves"). Then there is conflict between the higher and lower controls of the bodily functions—there are opposing influences with accompanying confusion. The cortex, however, can check only those bodily functions which are normally under voluntary control. That point I would emphasize. Just as the cortex cannot cause, so likewise it cannot prevent those stormy processes of the thalamus that increase the blood sugar, accelerate the heart, stop digestion, or produce the other disturbances characteristic of great excitement. When an emotion is repressed, therefore, it is repressed only in its external manifestations. There is evidence, to be sure, that when the external manifestations are maximal, the internal turmoil is also maximal[2]; and it is probable that cortical control of the outward display of excitement results in less internal disturbance than would accompany free expression. Nevertheless in a conflict between the cortical government and the activities of the thalamic centers the ungovernable internal manifestations might be intense.

The second point is related to evidence that states of consciousness are associated only with the cortical neurones. Certainly we are unaware of the numerous and complicated reflexes which determine bodily posture or the size of the pupil, for example, although these reflexes are regulated in the brain-stem. It follows that the neural mechanisms for the primitive emotions, active in the basal ganglia, are likewise probably not directly associated with consciousness. This consideration explains, I conceive, some of the most characteristic features of emotional experience. The disturbance in extra-conscious parts boils up into the realm of the conscious. Therefore, we have emotional "sei-

zures"; we may laugh, weep or rage "uncontrollably"; we feel as if "possessed"; what we do in the stress of excitement is "surprising" or "shocking"—something "surges up within us" and our actions seem no longer our own. These common bywords are explicable in terms of a sudden and powerful dominance of the bodily forces by subcortical neurones, i. e., neurones whose activity is not immediately attended by conscious states. Under favoring circumstances, with only a momentary lifting of the normal inhibitory check, these lower neurones capture the machinery of action and drive it violently into one or another of its variegated patterns.

I have now reviewed the evidence that the thalamic region, when freed from cortical control, is capable of elaborate independent activity of a stereotyped character; that when it acts it produces the typical reaction patterns in posture, expression and visceral responses that characterize various strong emotions; and that the activity of the thalamus occasions the feelings of excitement or depression which we experience during an emotional disturbance. Now the question arises, how are these considerations related to practical affairs? How do the processes going on deep down in the old part of the brain affect the workings of the body? To show how events in the thalamus can profoundly disarrange the nice adjustments of the normal organism, I shall cite some illustrative cases. I am sure that they will not seem unusual or improbable to many of you.

First, with regard to digestive functions. As stated earlier, my interest in the effects of emotions in the organism began with observations on the abolition of gastric peristalsis during excitement. Elsewhere[9] I have described instances of total stoppage not only of the mechanical action of the canal, but also of the work of the digestive glands, in consequence of emotional stresses. An evening's meal may remain undigested all night in the stomach if there is persistent worry during the period. The saliva, the gastric and the pancreatic juices all may be stopped by fear. The whole digestive process, which is subject to check by the sympathetic system, may be profoundly disarranged by anxiety and distress—the minor aspects of fear. McLester[10] has estimated that one-third of the patients with disorders of the alimentary tract are suffering because of lack of emotional balance. Alvarez[11] cites a case of persistent vomiting which started when an income tax collector threatened punishment if a discrepancy in the tax statement was not explained, and which ceased as soon as Alvarez himself went to the collector, as a therapeutic measure, and straightened out the difficulty. The natural processes of the alimentary canal are fundamental to all other functions of the body. Any disturbance of normal peristalsis, segmentation, and secretion of the digestive fluids may have widespread ill effects in the organism. Cabot[12] has recorded an instance of fracture of the leg which failed to unite. Investigation showed that the patient was fearful lest his family was suffering while he was absent at the hospital, i. e., the anxiety resulted in loss of desire for food (absence of hunger contractions of the stomach), that resulted in impaired nutrition, and that in turn led to such impairment of the reparative processes that the bone fragments were not welded together. Assurance that his family was well and happy, and being cared for, quickly altered the patient's condition; he ceased worrying, thereupon began to eat heartily and gain in nutrition, and then his broken bones began to knit.

The cardiovascular system, like the digestive system, is under the influence of the sympathetic nerves, but instead of being depressed or inhibited, it is stimulated by them. The excitement which stops gastric digestion makes the heart beat more rapidly and raises blood pressure by contracting the blood vessels. During the War there appeared not infrequently cases of "disorderly action of the heart" or, as it was sometimes called, "soldier's heart." The slightest excitement or perturbation would send the pulse bounding at a high rate (130 to 150 beats per minute). The general physical and nervous condition of the victims of this disturbance—their anxious faces, their troubled eyes, the drawn lines about the mouth, their trembling—was such as to make reasonable the view that the stresses of the war had become intolerable and had resulted in such sensitizing of the sympathetic control of the heart that even mild stimulation produced extreme effects[13]. The mechanism by which emotion may bring about such sensitizing is illustrated in a case reported by Foster[14]:—

> A wife, who was free from any cardiac disorder, saw her husband walking arm in arm with a strange woman and acting in such a way as to rouse jealousy and suspicion. Profoundly stirred by the incident the wife hastened home and remained there several days. She then began to fear going out lest she might meet her husband with her rival. After days of wretchedness she was persuaded by a friend to venture forth, "probably in a state of abject terror," as Foster remarks, but she had not gone far when she ran back to her home. Then she noted that her heart was thumping hard, that she had a sense of oppression in her chest and a choking sensation. Later attempts to go outdoors produced the same alarming symptoms. She began to feel that she might die on the street if she went out. There was no organic disease of the heart, and yet slight effort as she moved from her home brought on acute distress.

The influence of excitement on arterial blood pressure may also be noted. The pressure is produced by the energy of the inflow of blood into the arteries and the resistance to the outflow from them. The sympathetic impulses, by speeding the heart rate and constricting the arterioles, raise the pressure by affecting positively both factors. Gallavardin and Haour[15]

have reported after a study of 100 cases that the first time the blood pressure is taken, and the subjects are, therefore, excited, the systolic level may be 25 to 35 millimeters higher than it is later. And Schrumpf[16] relates an instance in which fear of a serious diagnosis raised the pressure 33 per cent., with prompt return to normal when reassurance was given. In extreme cases of pleasure, anger or fright a rise of 90 millimeters of mercury may occur. It is clear that patients suffering from hypertension and senile impairment of the circulatory system should avoid conditions and obligations which are likely to cause excitement.

Another effect readily produced by sympathetic impulses is the increase of blood sugar. The influence of emotional disturbance in bringing about a hyperglycemia in men subjected to the intense stresses of competitive sports or critical examinations I have pointed out elsewhere[1]. The same phenomenon has been observed in diabetic patients, probably because the sugar excretion is watched more closely in such persons. Woodyatt[17] cites the following from among many similar experiences in his practice:—

A man of 65 years, a diabetic, was in the hospital on a quantitative diet and with a small dose of insulin daily was passing a sugar-free urine. Suddenly one day, without any change in regimen, he secreted 43 grams of sugar. And on another day he secreted 76 grams and developed a mild acidosis; the glycosuria, therefore, could not have been due merely to the taking of extra food. A careful checking of all the circumstances and tests proved that there was no error of technique; nor did examination reveal any evidence of intercurrent physical disease. It was found, however, that the patient had received news which led him to fear that the corporation in which he had been an officer for more than 20 years had taken steps to retire him. That was the occasion for his disturbed sugar metabolism.

As Woodyatt remarks, "It is interesting to be able to measure the power of emotion in terms so tangible as ounces of sugar. *The power of emotions to produce physical alterations of the body* does not seem unreal under these conditions."

There is evidence that violent emotional disturbance can produce profound effects on the organism through influences on the thyroid gland. Marañon[18] has collected an extensive series of cases of hyperthyroidism brought on by stressful experiences during the Great War. Recently Emerson[19] has reported some striking instances of hyperthyroidism which followed intensely affective scenes in the lives of the patients.

One was a married woman who had had two illegitimate children and whose husband committed suicide in her presence as a rebuke to her manner of living. Thereupon she dropped to the floor and exhausted herself in shrieking. At once she had a sense of constriction of her throat and was troubled with difficulty in swallowing; the thyroid gland enlarged and six weeks after the incident she had a metabolism 65 per cent. above normal. Later troubles of an exacting character were associated with the development of high blood sugar and a high arterial pressure.

Another case. A man of twenty years had a quarrel with his fiancée. She, pretending to commit suicide, had in his presence swallowed some pills and fallen down screaming. The man departed hastily. Within a week he was suffering from swelling of the neck and nervousness. When he appeared at the hospital four months later he had lost weight, he presented a large goitre over which a definite thrill could be felt, and his basal metabolism was up 24 per cent. above the normal level.

A third case was that of a married woman who had seen her husband kill his two brothers. The husband bitterly reproached her for not coming to his defense at the trial. A week after the trial a goitre became evident and reached a large size in seven days. When she came to the hospital a few months later, the goitre was huge, it pulsated visibly, had a palpable thrill and was causing an oppressive sense of suffocation. There was pronounced exophthalmus with marked tremor and restlessness. The basal metabolism varied from +40 to +117 per cent.

There are other emotional effects on bodily functions which might be mentioned, such as disorders of menstruation[20], emptying of the bladder[21], secretion of milk[22], discharge of adrenin[23], altered coagulability of the blood[24], increase in the number of red corpuscles[25], and others. Enough instances have been given, however, to show that there are effects wrought on the organs innervated by the sympathetic nervous system—glands both of external and internal secretion and parts supplied with smooth muscle—that are just as real as the effects which are produced when the biceps is used to lift a weight. A remarkable difference lies in the level of the nervous control of these two effects. Whereas the biceps is usually managed from the cortex, the viscera are managed from the diencephalon. Whereas the biceps is under "voluntary" control, the viscera are not under that control, but are influenced favorably or unfavorably by processes associated with feelings and emotions. Although the neural center for emotional expression is subcortical indeed, is low in the brain-stem—yet cortical processes are involved in the total reaction to a situation which evokes strong feelings. We might be frightened by a real bear, but not by a stuffed bear. The discrimination between the two is made by the cortex. How may this relation between cortex and thalamus be interpreted in physiological terms?

Earlier I have pointed out that an emotional reaction has many of the characteristics of a reflex response. To evoke a reflex an appropriate stimulus must be applied; an irritant in the larynx produces coughing, food in the mouth calls forth a flow of saliva. Similarly with the emotional expressions. Watson[26] has studied new-born babies and has found that from the beginning loud sounds and also indications of loss of support are the natural stimuli for the

reaction of fear. Limitation or hampering of the freedom of bodily movement is from the beginning the natural stimulus for rage.

Agents other than the natural stimuli, however, can easily be made to set a reflex in action if only they are closely associated with the natural stimuli. Thus if a red light is flashed repeatedly at the same time that food is placed in the mouth, the red light will itself, alone, become as effective as the food in causing a salivary discharge. The indifferent stimulus, the red light, is then called the conditioned stimulus and the reflex salivary secretion, under the circumstances, a conditioned reflex. All sorts of ordinarily indifferent external agents—not only a light, but a sound, a shape, a contact, an odor, indeed *anything* that will influence a sense organ—may be made into an effective stimulus by close association in time with the normally effective stimulus. Thus objects and events in the world about us are constantly acquiring new significance for our reactions. All the processes of conditioning are carried on in the cerebral cortex. These facts, which have been studied in great detail and most instructively by the Russian physiologist, Pavlov[27], have pertinence for the explanation of emotional behavior.

Our emotional reflexes, like the salivary reflex, become complicated by the conditioning of indifferent stimuli. A white rat shown to a baby causes the baby to reach for it and to play with it; there is no fear. Then the rat is presented repeatedly but at the same time a loud sound is made by striking a steel bar. The rat thus becomes a conditioned stimulus for the fear reaction produced by the loud sound, and thereafter, when the rat is shown, the baby cries and turns away. He is now afraid of the rat not because it is a rat but because it has become the signal and symbol of something fearful—the loud sound. In such ways as this the indifferent circumstances of an emotional disturbance become conditioned stimuli or signals for renewal of the disturbance. The wife who saw her husband paying attention to a strange woman on the street had an intense emotional experience which was renewed, not by seeing again the errant husband and his distressing companion, but by going into the street! Thus by extended associations emotional responses become subjected to more and more involved conditioned stimuli, until great complexity and intricacy of affective behavior result.

In the foregoing discussion I have purposely emphasized the physiological mechanisms of emotional disturbances, and for two main reasons. First, I wished to show that these remarkable perturbations could be described in terms of neurone processes. And again, I wished to persuade you that these interesting phenomena should not be set aside as mystical events occurring in the realm of the "psyche," but rather should be regarded as movements and inhibitions and disturbances in the body which properly fall within the province of the physician.

Probably a physiologist is venturing too far if he attempts to suggest practical modes of treatment. And yet in what I have presented to you there are physiological implications which have practical bearings on the care of patients who have been or are being profoundly disturbed by emotional experience.

First, there is the importance of early treatment. We are all acquainted with the readiness with which habits are established in the nervous system by frequently repeating an act. Every time the nerve impulses traverse a given course they make easier the passage of later impulses. Thus habitual emotional expressions, both in the facies and in the viscera, may become fixed and deep-set in the neural organization, just as the complicated adjustments of swimming, skating or bicycle riding become inwrought during our later years by repeated practice. It is clear that so far as possible emotional habit-reactions should be prevented by prompt treatment.

As we have seen, the cortex has no direct control over the functions of the viscera. It is useless, therefore, to try to check a racing heart or to lower a high blood pressure, or to renew the activities of an inhibited digestive system by a coldly reasoned demand for different behavior. The man whose broken bone failed to knit because he was fearful about his family's welfare could not be *argued* out of his fear; the fear left him when he learned that his family was actually comfortable. The cortex, which is concerned with analysis of the outer world, should not, therefore, be the sole means by which treatment is attempted; the *occasion* for worries, anxieties, conflicts, hatreds, resentments, and other forms of fear and anger, which affect the thalamic centers, must be removed. In short, the factors in the whole situation which are the source of strong feeling must be discovered and either explained away or eliminated.

Although the cortex has no direct control over the viscera, it has indirect control—we can walk into danger and have a thrill, though we cannot have a thrill by merely resolving to have one. Similarly we can often avoid the circumstances which rouse fear or rage or disgust and their attendant visceral turmoil—we need not go near the agitating spot.

Again, when the reason for the perturbation is not clear, it can sometimes be found by careful enquiry or analysis. It is an interesting fact that a full explanation of the way in which the trouble has been caused will not infrequently suffice to remove the trouble, promptly and completely.

Finally, a word of warning may not be out of place. If an objective cause for a patient's

complaint is not found, nothing is easier than to attribute the difficulty to nervous factors. There is danger, when one emphasizes the importance of nervous factors as disturbers of the bodily peace, that one may be understood as minimizing the need of search for a gross pathology. Nothing could be farther from my intention. The assumption that emotional agencies are causing mischief in the organism should be a last resort—an explanation which is offered only after every effort has been made to find another explanation. And even when the cause is ascribed to fear or rage or some other strong feeling, proof for that conclusion should be carefully sought both at the source of the trouble and in the effect of appropriate therapy. Nor should the possibility be overlooked that along with profound emotional disturbance there will be discovered a demonstrable lesion. The two conditions, the altered structure of some organ and the altered function of the nervous system, may be causally related, and may have to be treated as a single disorder. Certain it is that only when they are both regarded as the perturbations of a single unity, the organism, will they be properly conceived and effectively treated.

I have tried to indicate the ways in which the functions of the body may be upset by the neural processes which are associated with emotions. I hope that I have convinced you that interest in this realm of medicine should not be relegated to cults, mental healers and the clergy. The doctor is properly concerned with the workings of the body and their disturbances, and he should have, therefore, a natural interest in the effects of emotional stress and in the modes of relieving it. The field has not been well cultivated. Much work still needs to be done in it. It offers to all kinds of medical practitioners many opportunities for useful studies. There is no more fascinating realm of medicine in which to conduct investigation. I heartily commend it to you.

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SOME UNPROVED IMPRESSIONS CONCERNING THE SUBJECT OF HEART DISEASE*

BY SAMUEL A. LEVINE, M.D.†

DURING prolonged and intimate observation of a great variety of patients suffering from heart disease, one cannot help but develop certain impressions as to the nature of the disease, entirely apart from the more orthodox conceptions that prevail. Some of these impressions in the natural course of time will turn out to be ill-founded. Others, however, it may be difficult to prove and yet they may serve as helpful working hypotheses. This is particularly true of those matters which require extensive statistical study for their complete elucidation. Good examples of the latter are the impressions that prevailed some time ago that rheumatic heart disease was definitely more common in the northeastern states of this country than in the southern. This has been quite well established more recently. The same has been true concerning the familial tendency of rheumatic fever or of rheumatic heart disease. This was thought by many to be true for some years before the work of Dr. St. Lawrence in New York quite definitely established the fact. It is the type of such impressions that I wish to discuss at this time.

We are all familar with the frequent difficulty in deciding whether certain children suffering from vague complaints really have rheumatic fever or not. It is quite clear that many of them need have no arthritis whatever and yet have rheumatic fever. We are often presented with the problem of interpreting chest pain, palpitation, the presence of a simple systolic murmur, or other vague symptoms like fatigueability or nervousness, apart from the more usual criteria of rheumatic infection. There are two features in many of these children that have proved helpful in deciding whether the problem is one of rheumatism or not. These are repeated epistaxis and attacks of painless nausea and vomiting. These symptoms have not seemed to bear any necessary relation to the more outspoken evidence of rheumatic infection, namely polyarthritis, or active chorea. At times, they occur with the onset of the initial disease and at other times there is a story that goes back for years of repeated nosebleeds or spells of vomiting, occurring during a time when the patient is apparently well, and finally, they may occur after the patient has recovered from the fulminating rheumatism, while back at school and in fair health. The exact relation of these symptoms to the underlying disease is obscure, but it is surprising how frequently they occur, certainly more so than can be explained on the basis of chance. As a practical matter, they have been valuable as secondary features in estimating whether some of the patients with atypical findings were suffering from rheumatic infection.

All of us have been greatly impressed with the importance of the familial factor in various kinds of heart disease. This is true, both of the rheumatic group of heart disease and of the degenerative type such as is found in hypertension or in coronary disease. It has impressed me in recent years that one may go still further and say that there are individuals who inherit the general vulnerability to vascular disease, whether infectious or degenerative. Too frequently to be accidental have I noted the presence of rheumatic heart disease in the children and coronary disease in the parents. There are many families in which the father has angina pectoris or has died of it and one or more of the children have rheumatic valve disease. It would require very extensive statistical study to prove the truth of this impression. It is obvious that if this is true, we cannot be dealing with a question of contact infection or inheritance of the same disease, for one is infectious and the other is degenerative. It must therefore be that a definite vulnerability of the vascular system exists that is transmitted.

The constitutional factors behind the development of morbid states have been called to our attention in the masterly work of Dr. Draper of this city. His teachings I have followed with great interest and have seen to be applicable in three different types of disease, namely pernicious anaemia, angina pectoris and to some extent the rheumatic infections. It is striking how many children with freckled faces and red hair come to heart clinics and how many stocky, well-set men have angina pectoris. Other characteristics of the anginal patient, it has seemed to me, have been round rather than flat forearms and in these individuals the skin fits tightly around the limbs and abdomen. All this leads me to the conclusion that the infectious agent producing rheumatic heart disease, which is very prevalent in this part of the country and is occurring more frequently than we recognize, runs a mild atypical course and produces heart or valvular damage in those with a vulnerable vascular system. This can explain many cases in which a child may have mild symptoms, slight fever, occasional aches, nausea and epistaxis, in which no permanent heart injury occurs. One may compare this conception of the frequency of rheumatic fever with the general opinion that anterior poliomyelitis is much commoner than is recognized and

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that only a small proportion develop the characteristic nerve lesions.

Another striking phenomenon that occurs in one of the important types of heart disease that needs explanation is the great frequency with which the identical blood vessel is involved in cases of coronary thrombosis. In 80-90% of the cases of coronary thrombosis, the left descending coronary artery is the site of the main injury. This apparent specificity or localization of the degenerative process is a phenomenon which deserves more attention than has been paid to it. It is, furthermore, quite remarkable how commonly a particular part of this vessel is involved. Frequently that portion of the left coronary artery which lies about one inch from its origin is the site of the lesion. The question that comes up is whether some individuals have a peculiar anatomical architecture of the heart or some queer curves in the course of the vessel which predisposes them to early sclerotic changes. If this were true, it would fit in logically with the great frequency with which we see early angina pectoris occurring in certain families. For, after all, anatomical structures with their peculiarities are the evident characteristics that we inherit.

To turn to a different aspect of heart disease that has puzzled me, there are certain incompatibilities between common conditions that are rather difficult to explain. I refer particularly to three matters. The first is the apparent antagonism between auricular fibrillation and angina pectoris. Some years ago I called attention to the great rarity of the two conditions existing together. Both auricular fibrillation and angina pectoris are so common that from a statistical point of view, apart from the fact that they are both heart disorders, one would expect to find numerous instances of their co-existence in the same patient. This, however, is not so—in fact the previous existence of persistent auricular fibrillation, even when compensation has been well established, is a real protection against the development of angina pectoris. I have no adequate explanation for this.

The second incompatibility, attention to which has been called by Dr. Libman and his associates, is the one that exists between congestive heart failure and fibrillation on the one hand and the development of subacute bacterial endocarditis on the other hand. One may go further and call attention to the fact that although mitral stenosis is the most common type of rheumatic valve disease, yet bacterial endocarditis more frequently develops in patients who have had aortic insufficiency than in those who have had mitral stenosis. This does not mean that the aortic valve is more commonly involved in subacute bacterial endocarditis than the mitral valve, but if the patient has evidence of mitral stenosis, he is distinctly less liable to develop subsequent bacterial endocarditis. It is those patients who either have aortic insufficiency, or who have a mitral systolic murmur without evidence of mitral stenosis, who are the most vulnerable in this regard. I wonder whether this question is linked up with the presence of scar tissue. The dense fibrous leaflets that are seen in mitral stenosis are not unlikely less fertile soil for the superadded infection by the streptococcus viridans. Most of the mitral valves that are seen at autopsy in cases of bacterial endocarditis give the appearance of having been in a fair state of health before the terminal infection developed. This would explain the rarity of subacute bacterial endocarditis in rheumatic patients who have persistent auricular fibrillation and in patients who have had congestive heart failure, for in both of these conditions the tissue of the mitral valve is apt to be dense and fibrous It would also explain the frequency in aortic insufficiency, for here congestive heart failure is a very late development and the span of life after it occurs in an aortic case is apt to be short. Because of the fact that auricular fibrillation in rheumatic heart disease is almost always associated with mitral stenosis and that congestive heart failure in a patient with mitral stenosis comes most commonly after auricular fibrillation has set in, it would not be expected that bacterial endocarditis would develop so frequently under these circumstances, if dense fibrous tissue is an inhibitory factor.

The third antagonism that seems to exist in certain types of heart patients is their comparative freedom from tuberculosis of the lungs. In the Peter Bent Brigham Hospital we have had only one proved case that had both pulmonary tuberculosis and mitral stenosis. Viewed from the other angle, I learn from the physicians in Saranac that cases of mitral stenosis are almost unheard of amongst their hundreds of tubercular patients. One might speculate and say that the mechanical factor of increased pulmonary pressure and consequent passive hyperaemia of the lungs militate against the development of pulmonary tuberculosis. But may it not also be true that fundamentally two different constitutional factors are involved, a vulnerability to rheumatic infections or to vascular disease and a predisposition to tuberculosis, and that the presence of the one means the absence of the other? For, even in cases of rheumatic aortic insufficiency where there need be no congestion of the lungs for many years, it has seemed that tuberculosis is rare. A further point in this connection is the great rarity of pulmonary tuberculosis in hypertensive individuals. Although this does occur, the frequency of both conditions in the same individual seems to be uncommon. Dr. Minor of Asheville who has had an extensive experience in the treatment of tuberculosis has stated that he practically never finds hypertension, and the converse of this is true in clinics where large numbers of patients with hypertension are observed.

When we turn to the prevention of heart disease, there has been considerable discussion concerning the rôle that foci of infection and the tonsils play as causative factors. These questions have by no means been satisfactorily answered. There is, however, one factor that has seemed to me to be of some importance and yet has received insufficient emphasis. I refer to the question of weight. We are generally accustomed to think of average weights in relation to age, height and sex, in advising our patients. It must be remembered that average figures are not necessarily the best and are made up of good, bad and indifferent elements. The optimum weight at one age may be above the average and at another age may be below the average. This I believe to be true in relation to heart disease. It is very rare indeed to see chorea in an obese child and uncommon to find a child who is overweight come down with the first attack of rheumatic fever. On the other hand, after the age of 35 or 40, thin individuals seem to be less likely to develop angina pectoris. In fact, I believe that to be well and below average weight at the age of 40 means a distinctly greater life expectancy. It follows from the above that in an attempt to prevent heart disease, we should try to have children above normal weight and adults after 40 below the standard figures.

A final subject that at present is receiving a great deal of attention and emphasis, I feel deserves discussion. I have reference to the relation between physical activity and cardio-vascular disease. There is an intensive wave of enthusiasm throughout the country for athletics, both in the young and in the old. There is simultaneously a nation-wide interest in the great prevalence of cardio-vascular disease. I have felt for a long time that the evidence in favor of the beneficial effect of athletics upon the circulation is very meagre. There has been great confusion in the minds of many between the production of a sense of well being and a feeling of fitness that follows athletics, and an improvement in the circulation. Certain muscles in the body may be strengthened by exercise, but I find no proof that the strength or health of the circulation is similarly improved.

From the point of view of longevity the most important factor in the body is the circulation. This part of our body is never at rest and is not subject to the deterioration that may come from disuse. Is it not logical to believe that the more it is used, the sooner it will wear out? It is evident to all physicians that when there is circulatory failure of the congestive type, physical activity aggravates the condition and rest improves it. It would be queer if the laws of nature were so fickle that the reverse of this were true, after the circulation in the same sick patient had already been improved. The most satisfactory criterion of the state of the circulation when the question of congestive heart failure is to be considered is the patient's ability to breathe. In measuring this, apart from the common methods of physical examination, the vital capacity of the lungs has served as a satisfactory guide. This has been well shown by Dr. Peabody and his coworkers to improve with rest. If exercise actually improves the strength of the circulation and the vital capacity of the lungs is an indication of this improvement, then it would follow that men in training would have abnormally high readings. In carrying out some observations on Marathon runners some years ago, on men who had been running long races for years and who were the most experienced Marathon runners in this country, I found no such general increase in the vital capacity of the lungs. Some were normal, some were below normal, and some were increased. Large numbers of our young men at schools and colleges are playing strenuous games like football and the older men at present are playing tennis and golf so enthusiastically that one may well wonder about the possible harm that the vascular system undergoes as a result of the added work. These matters so far as I know have not been subjected to satisfactory statistical proof, but it is very impressive to me how many of the vigorous athletic men are dying of coronary disease. Is it merely accidental or is it cause and effect?

THE FLUOROSCOPIC REMOVAL OF METALLIC FOREIGN BODIES IN THE BRONCHI*

BY D. CAMPBELL SMYTH, M.D.†

IN the presentation of this paper, I must admit at the very outset that there is in it very little that is new. Fluoroscopic aid in the removal of opaque foreign bodies from the air passages has been used more or less for a number of years. I can remember, more than fourteen years ago, one patient with a small screw nail in a terminal bronchus, which was successfully removed by this method. However, at that time there was not nearly the precision and accuracy in the method that there is today.

This paper is more or less a plea to use fluoroscopy early rather then leave it as a last resort procedure. In my opinion it should be employed early in selected cases, as the danger of removal by this method has been exaggerated.

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In fact, I doubt very much if the mortality is at all raised, if the work is gently and carefully done, and the exact location of the foreign body thoroughly studied beforehand by roentgenogram and fluoroscopy. The higher mortality by this method is, in my opinion, largely accounted for by the fact that it is used in the more desperate cases, and often left as a last resort in those cases where it would have been successful after one try by direct vision had failed.

Of course, I admit that the ideal way of removing a foreign body is by direct vision, but as I just stated, when one real attempt by a competent bronchoscopist by direct vision has failed, or when it is known previously that the intruder is lodged in a terminal bronchus, especially if it has resided there for a very considerable time with probable granulations in, or even cicatricial stenosis of, the affected bronchus, fluoroscopic removal should be resorted to rather than blind removal by using the end of a grasping forceps as a probe, and attempting to seize the foreign body in the dark. While, then, removal by direct vision, as I stated, is the method *par excellence* when this is not possible, we have at hand fluoroscopic vision, the nearest approach to real vision. Therefore, my inclination is to regard the fluoroscope as a first aid. With its employment the danger of grasping lung tissue and tearing it is certainly much slighter than if the foreign body is removed by tactile sensation alone, for by this method we still have our tactile sensation plus fluoroscopic vision.

The operator must be gentle in his manipulations, avoiding trauma and at the same time, if he has an expert fluoroscopist, he will be told a great deal about the manipulation of his forceps and their relation to the foreign body. He must be prepared, before he starts, to completely subordinate himself to the fluoroscopist and to follow explicitly his directions, even to the point of letting go of the foreign body when told to do so, in order that a more favorable grasp for safe removal be made. This has occurred in one or two of our cases, notably in the case of a tack, which I grasped at about the middle; removing it in this way would have perforated the bronchus. I was told to push it down farther to dislodge the point, and was able immediately to grasp it in such a way as to make removal safe.

During the last two years at the Massachusetts Eye and Ear Infirmary fluoroscopic removal has been resorted to in eight patients, once by Dr. Green, once by Dr. Herman, once by Dr. Poirier, twice by Dr. Mosher, and three times by myself. In six of these patients one or more attempts by direct vision had failed, while in the seventh, where the foreign body had been lodged for over thirty-one years and was known to be in a dorsal terminal bronchus with partial stenosis of that bronchus, it was used as the method of first choice, with success. In the eighth patient the foreign body, a large dental bridge, was grasped by direct vision and extracted under fluoroscopic control. The fluoroscopist in all those cases was Dr. A. S. MacMillan, Massachusetts Eye and Ear Infirmary.

As to the choice of anesthesia, we do not confine ourselves to any one, but rather try and suit it to the individual patient. In bronchoscopy for lung mapping and treatments we use local anesthesia combined with morphine. We do the same in most instances where the foreign body has been in the lung but a short time. Where we know the removal is going to be difficult, and especially if the foreign body has been lodged for a long time, we use general anesthesia as first choice, usually ether, either rectal or by mouth.

One of the great drawbacks of general anesthesia has been the difficulty of grasping foreign bodies well down at the base of the lung, due to the excursions of the foreign body with the diaphragm movements, sometimes this movement being as much as one and one-half to two inches. Under fluoroscopic removal this excursion bothered the operator and fluoroscopist a great deal until I found that by holding the diaphragm on the side of the foreign body, it was possible to keep it stationary, or nearly so, thus making grasping of the intruder much more accurate. It is equivalent to telling a patient to hold his breath, when using local anesthesia.

The time required for removal of the foreign bodies in these cases varied from one to forty-five minutes. This I do not regard as a constant important factor. In young children the element of time is important, of course, especially with non-opaque foreign bodies where the children, when they come to us, are desperately ill. In adults with any acute condition present it is again a factor, but in adults where the foreign body has been lodged for a long time and no acute condition is present, as much time as would be consumed for any major surgical operation may be taken without reaction of a serious sort, provided the operator is careful to avoid all traumatism and his every manipulation is gentle.

I have seen less reaction to prolonged bronchoscopy in such cases than in the very short bronchoscopies under local anesthesia in lung abscesses. Naturally, of course, it goes without saying that once the bronchoscope is introduced, it should not be removed, neither should its excursions in and out be any more than absolutely necessary.

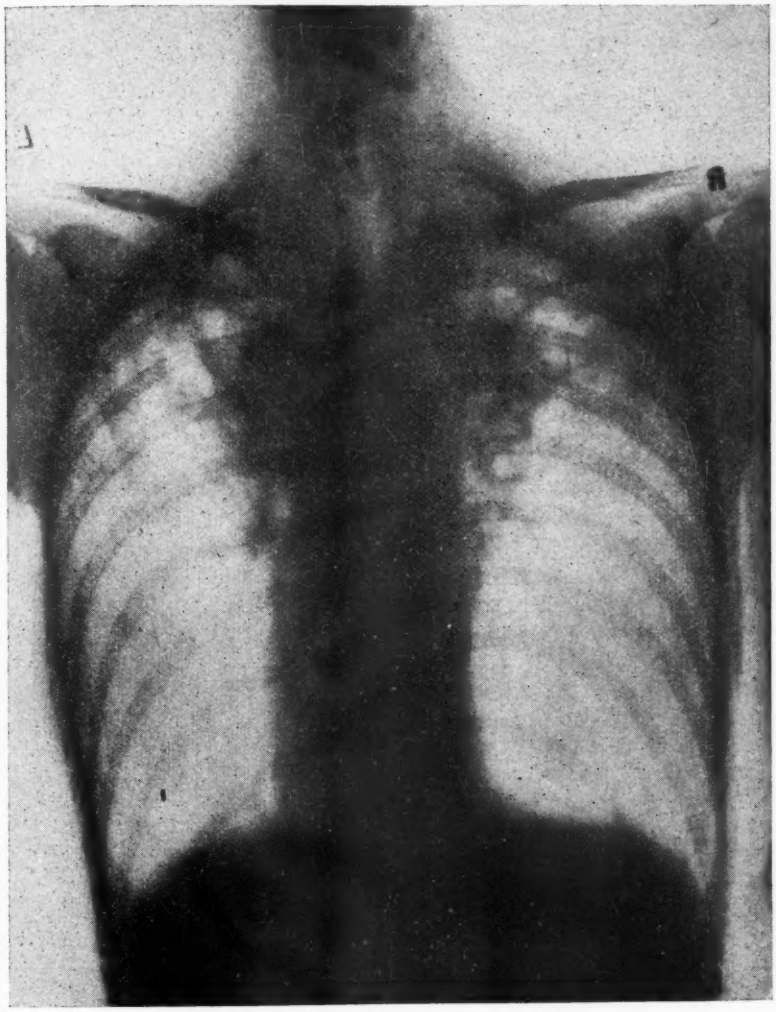
CASE HISTORIES

CASE I. M. C. Aet. 30.

It may seem ridiculous to require fluoroscopy for the removal of a foreign body from the upper end of the oesophagus, but in this patient it was a decided aid.

The patient, a young woman, under treatment for active tuberculosis, was in the habit of having a glass of milk at her bedside to drink during the night. One night, as she drank it, she felt something stick in her throat. The pain persisted until I saw her three days later. X-ray examination showed a very small open safety-pin at the entrance to the oesophagus.

Under local anesthesia a thorough search was made among the swollen folds at the upper end of the oesophagus. Finally, deciding the foreign body might have been dislodged and pushed along, a large Mosher oesophagoscope was passed down the oesophagus, but no foreign body was seen. Another X-ray showed the foreign body in the same location. The following day the patient was placed on the fluoroscopic table and with a two-plane fluoroscope the pin was very quickly located and removed from among the oedematous folds.



CASE I

Small inverted safety pin hidden among the folds of the upper end of the end of the Oesophagus.

CASE II. E. M. Aet. 31.

Inhaled a shawl pin eleven days ago.

X-ray showed that the pin had gone down the right bronchus.

February 23, 1926. An unsuccessful attempt was made to remove the foreign body by direct vision.

X-ray following operation showed the pin farther down, evidently deep in a terminal bronchus.

Three days later the pin was successfully removed with fluoroscopic aid. No reaction followed the operation.

CASE III. N. H. Aet. 35.

History of frequent attacks of bronchitis for two years, following a tooth extraction.

X-ray showed what proved to be a flat amalgam filling, in the right bronchus.

An attempt made to remove the foreign body by direct vision was unsuccessful. The foreign body was neither seen nor felt.

Several days later the foreign body was located and removed, under fluoroscopic control.

No reaction followed the operation.

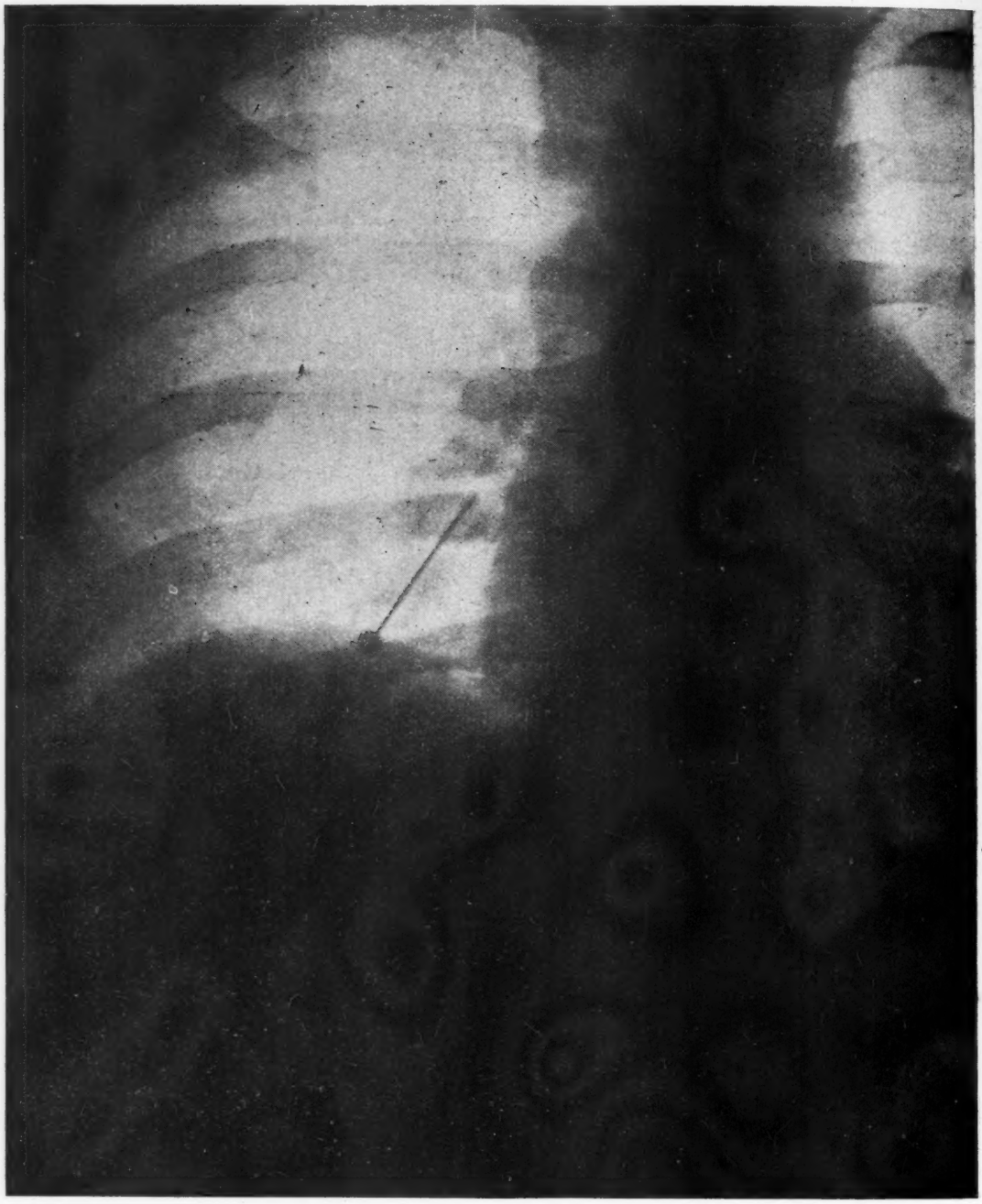
CASE IV. J. K. Aet. 9.

Patient came to the hospital with a history of tonsillectomy a few days before. At the operation two teeth were lost.

X-ray at that time was said to show one in the intestinal tract, and the other in the right bronchus about the opening of the middle lobe.

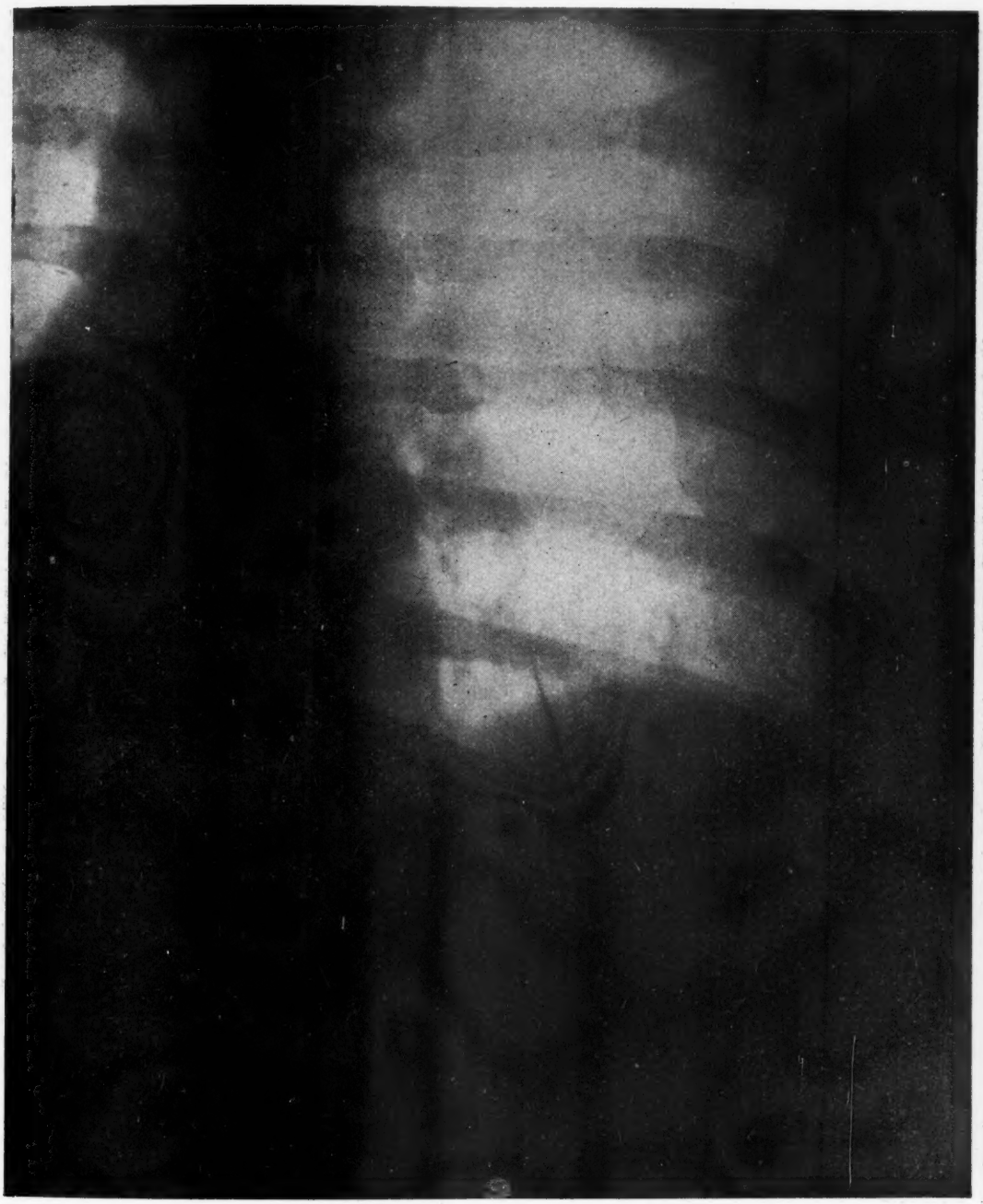
An unsuccessful attempt was made to remove the foreign body by direct vision.

The foreign body was located and removed from the middle lobe bronchus several days later under fluoroscopic control.



CASE II

Showing a pin in the right main bronchus before an attempt was made to remove it by direct vision.



CASE II

Showing the pin further down in terminal bronchus after the attempt had been made to remove it by direct vision. It was easily removed from this location with the aid of the fluoroscope.

CASE II
A lateral view of the same patient.

CASE V. M. S. Aet. 5.

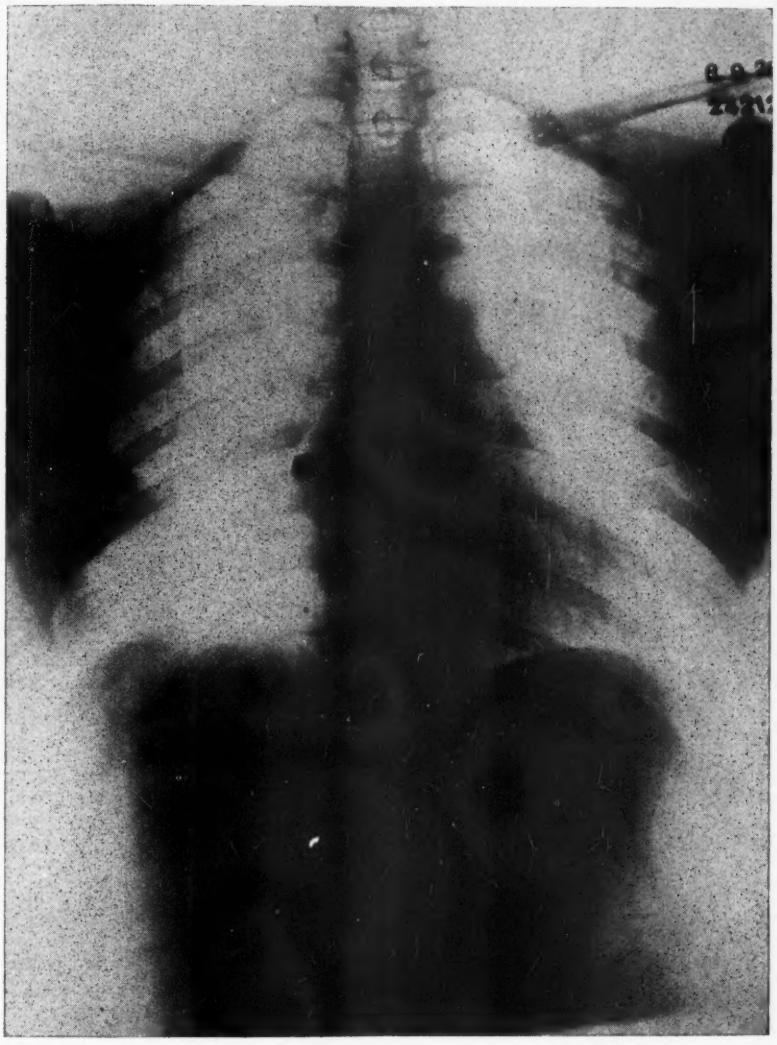
History of cough and loss of weight for three months. No history of inhalation of foreign body. Temperature on admission was 101°.

Child had a loose, non-productive cough. X-ray showed an upholstery tack in the right bronchus with no air in the lower part of the lung.

July 11, 1926. Attempt made by direct bronchoscopy to remove foreign body failed.

July 15, 1926. Attempt by fluoroscopy failed. Tack located in right middle lobe bronchus.

July 28, 1926. Tack removed by fluoroscopy (vertical screen) at side of child.



CASE III

Showing a flat amalgam filling which proved to be buried in the wall of the right main bronchus. Inhalation was two years previous. First attempt at removal by direct vision was unsuccessful. Several days later it was easily removed under fluoroscopic control.

The foreign body was first grasped at the centre of the shaft. Tactile sensation showed resistance. The fluoroscopist advised moving the foreign body downward. The grasp was then released and the point of the tack immediately picked up by following the fluoroscopist's directions.

Numerous plates were taken at intervals to show the condition of the lung.

CASE VI. R. T. Aet. 2.

Carpet tack in right bronchus for three weeks.

While being extracted, the tack was lost at the glottis. It was immediately located by the fluoroscopist in the left bronchus, and quickly removed from its new location.

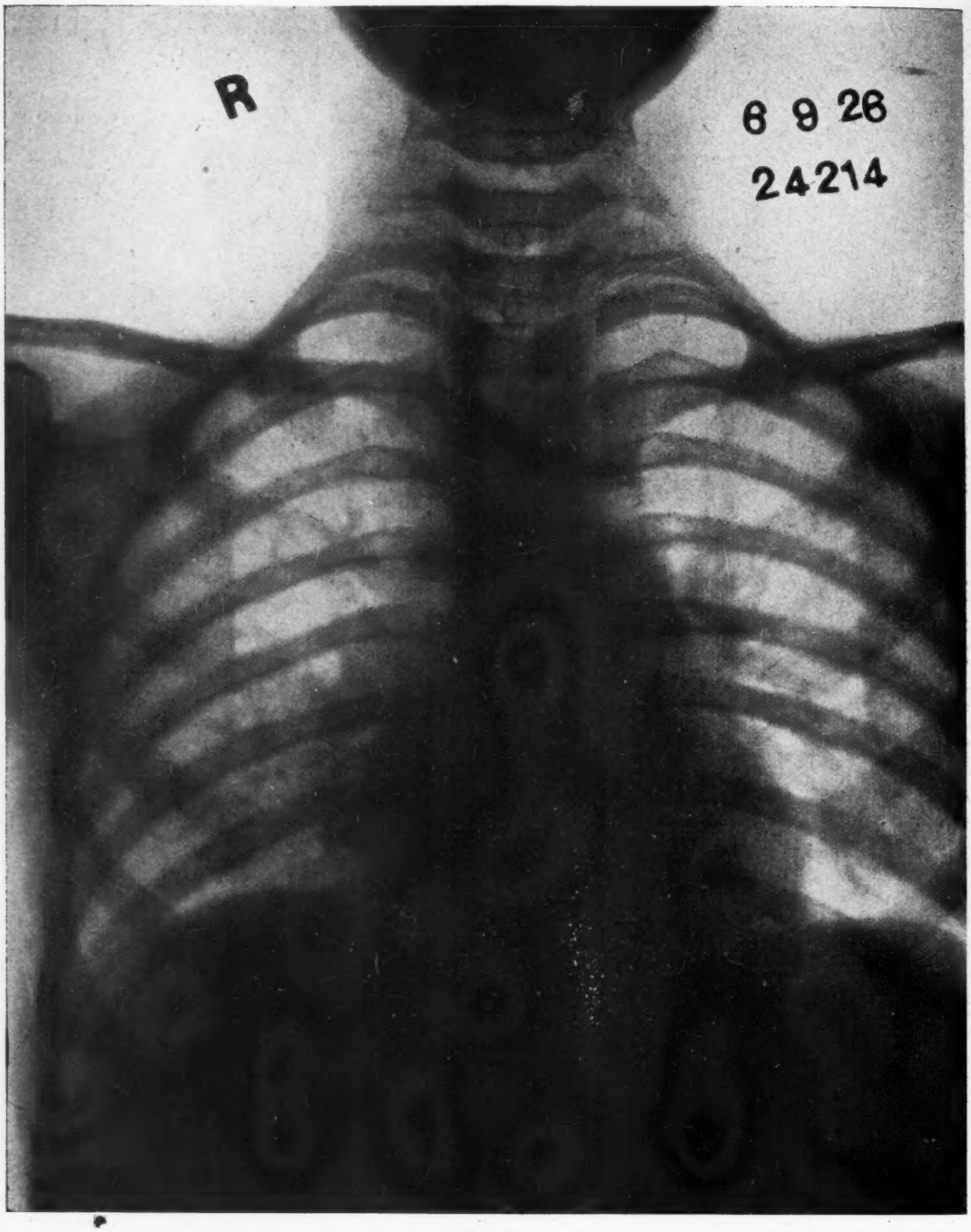
The child had an acute bronchitis at the time, and the quick location of the foreign body in the left bronchus by fluoroscopy was a decided aid.

CASE VII. S. M. Aet. 39.

This patient had a history of inhaling a gold-headed scarf pin over thirty-one years ago. During her early childhood the case was repeatedly diagnosed as probable tuberculosis on account of her persistent cough with bloody sputum.

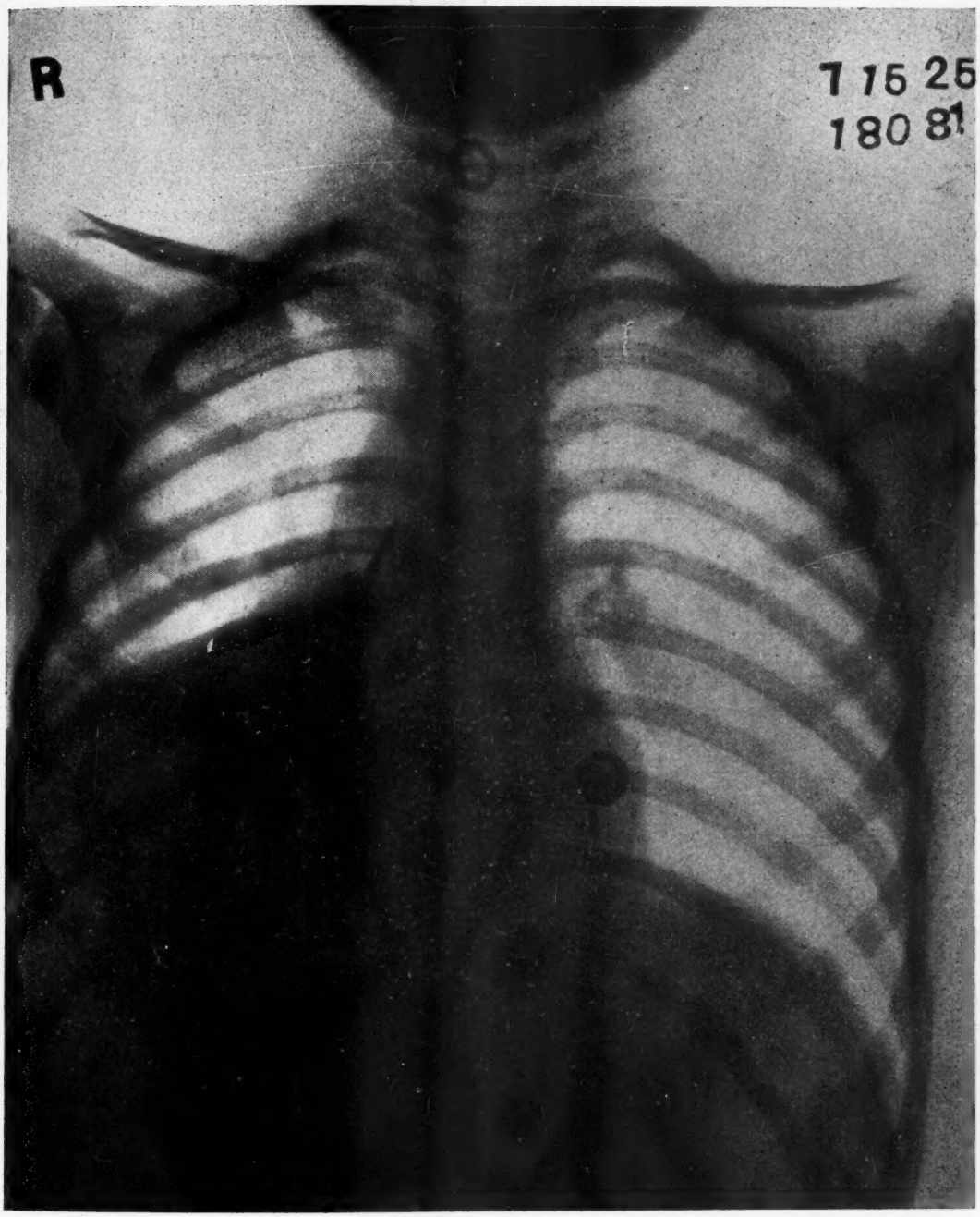
Ten years ago she was seen by an expert on tuberculosis in Boston, and at that time she had the first X-ray plate of her chest taken. It revealed the unsuspected foreign body at the base of the right lung. Her mother then remembered the incident of her having "swallowed" a scarf pin when she was a child. At this time (that is ten years ago) she was seen by a bronchoscopist in consultation with the internist. They both agreed that it was inadvisable to try and remove the foreign body.

I first saw her last July. She gave a history of at present having an almost constant cough, especially in the winter, at times raising a fairly large amount of sputum and then going a number of weeks



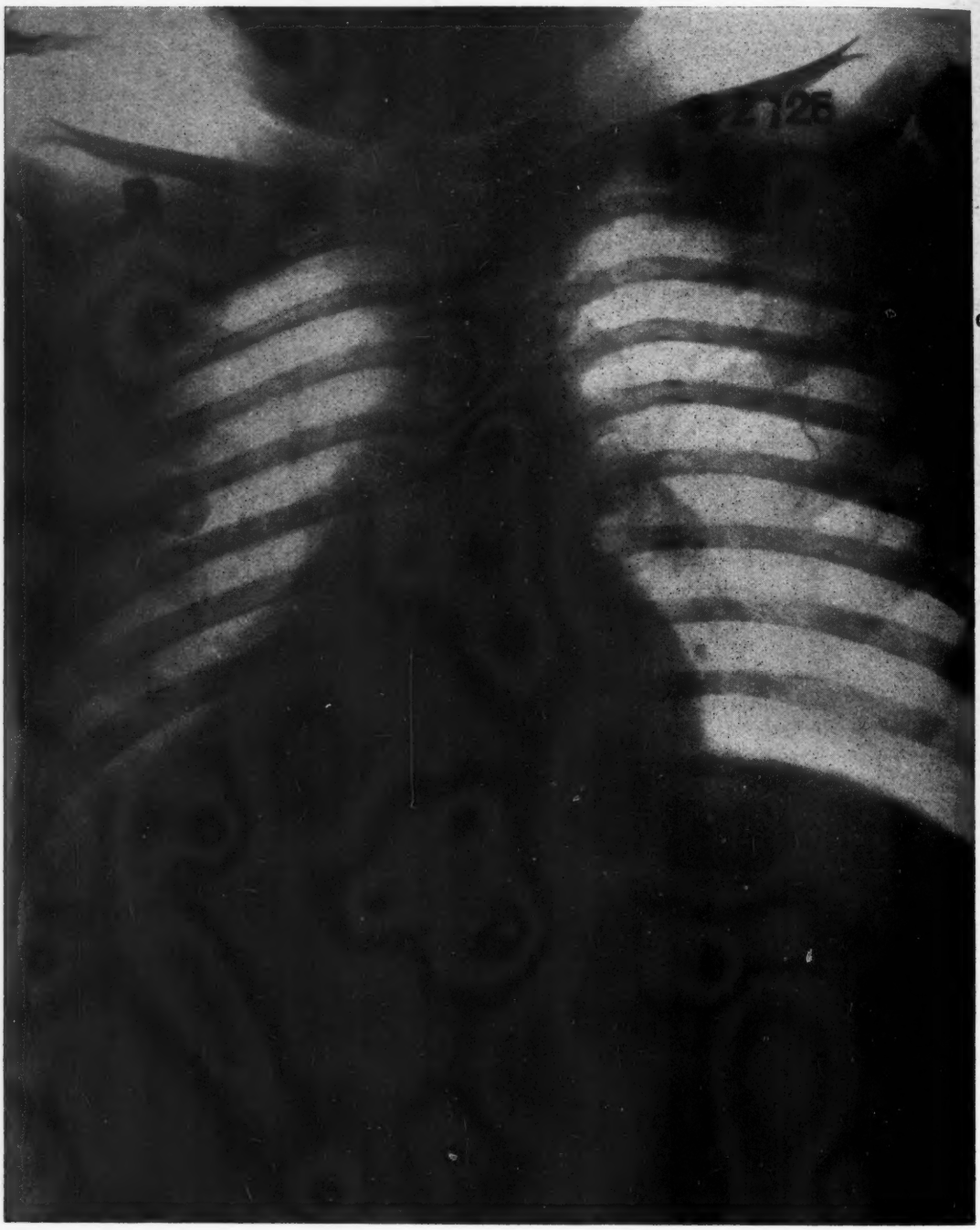
CASE IV

Showing a small desidious tooth in the right middle lobe bronchus of a child 9 years old, inhaled during tonsillectomy. Removed successfully with the aid of fluoroscope after one attempt by direct vision had failed.



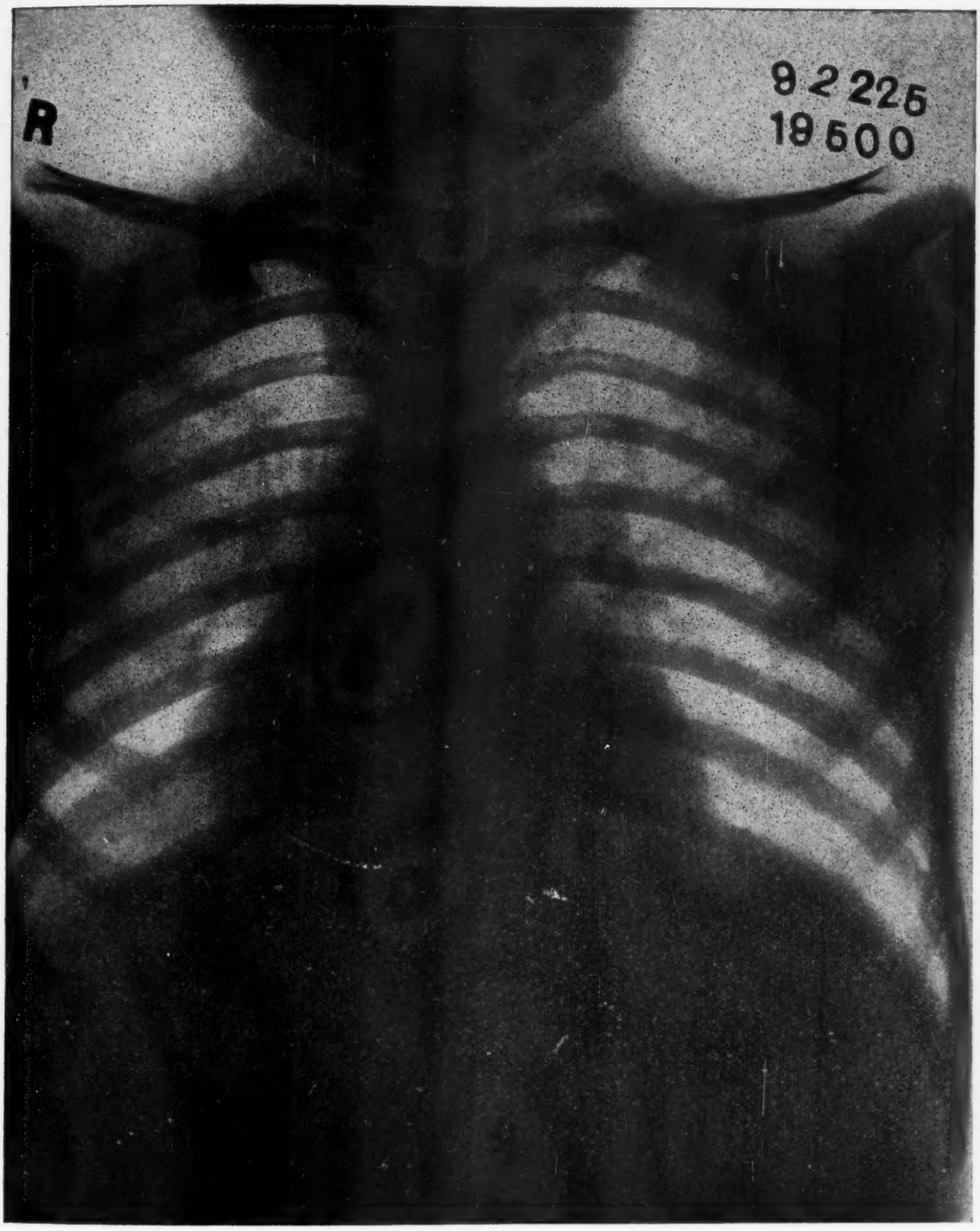
CASE V

Showing upholstery tack in the right middle lobe bronchus in a patient 5 years of age. The history pointed to inhalation about 3 months before. This foreign body was removed by fluoroscopy, using a vertical screen at the side of the patient.



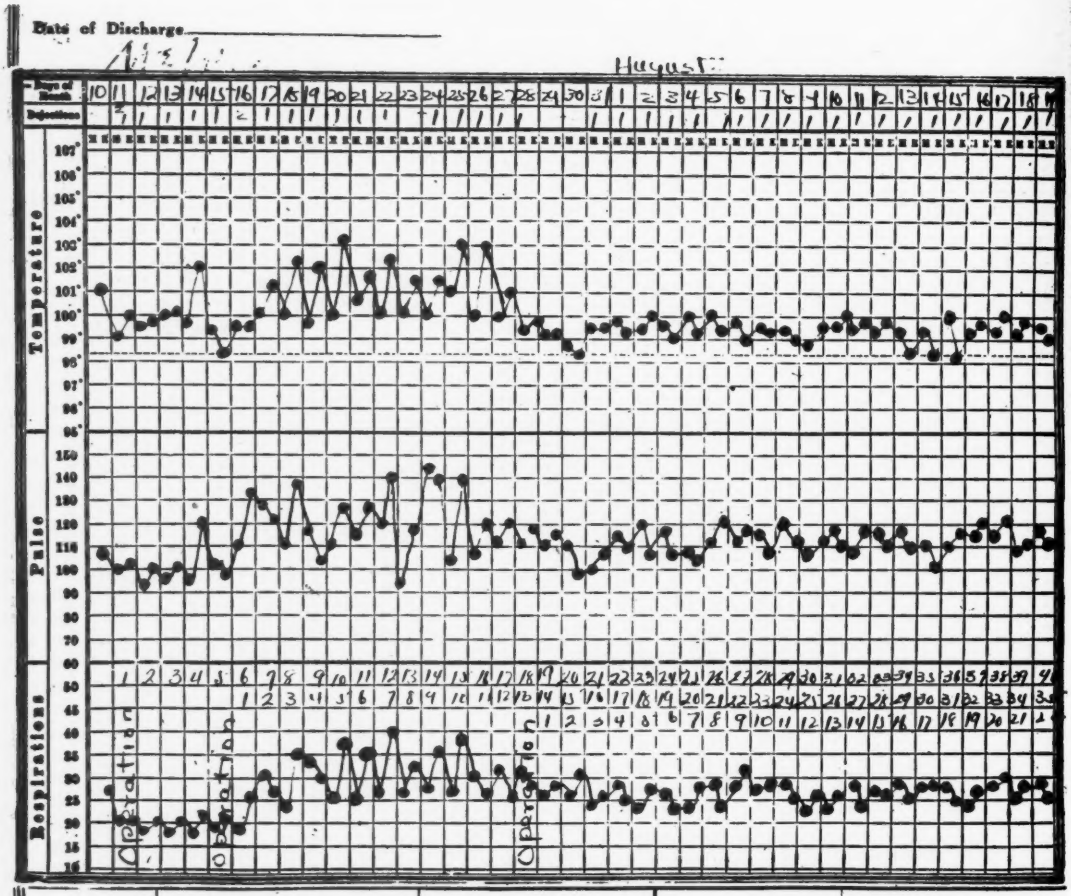
CASE V

Showing the chest about 4 weeks later. There is still considerable consolidation at the right base and the heart is still pulled over.



CASE V

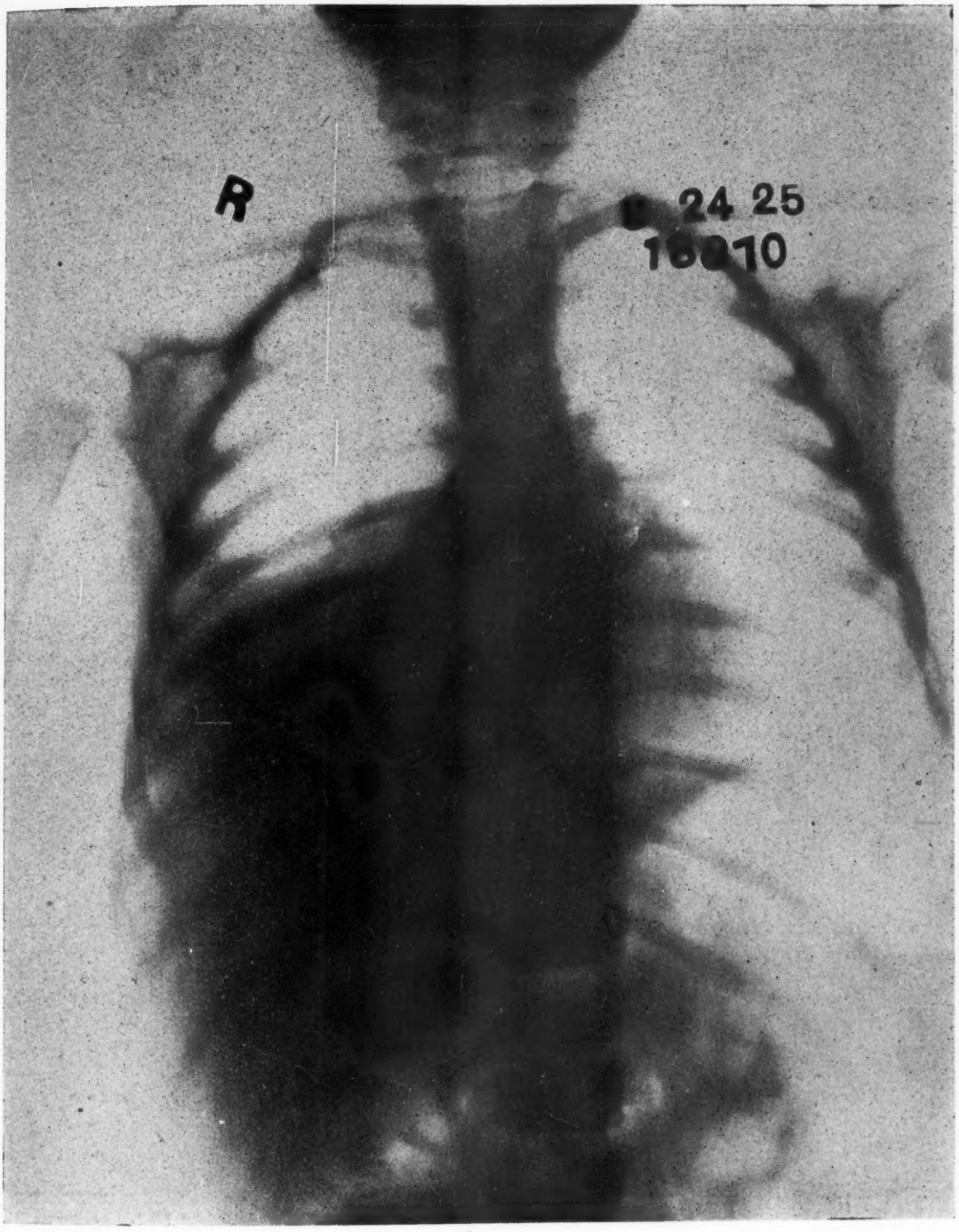
Another plate 2 months later showing the lung apparently restored to normal with the heart in normal position.



DATE	BLOOD CORPUSCLE COUNTS	H G B	STAINED SPECIMENS
7/19/25	W.B.C. 22,400		
	R.B.C. 4,080,000	Abg 70%	Not remarkable —

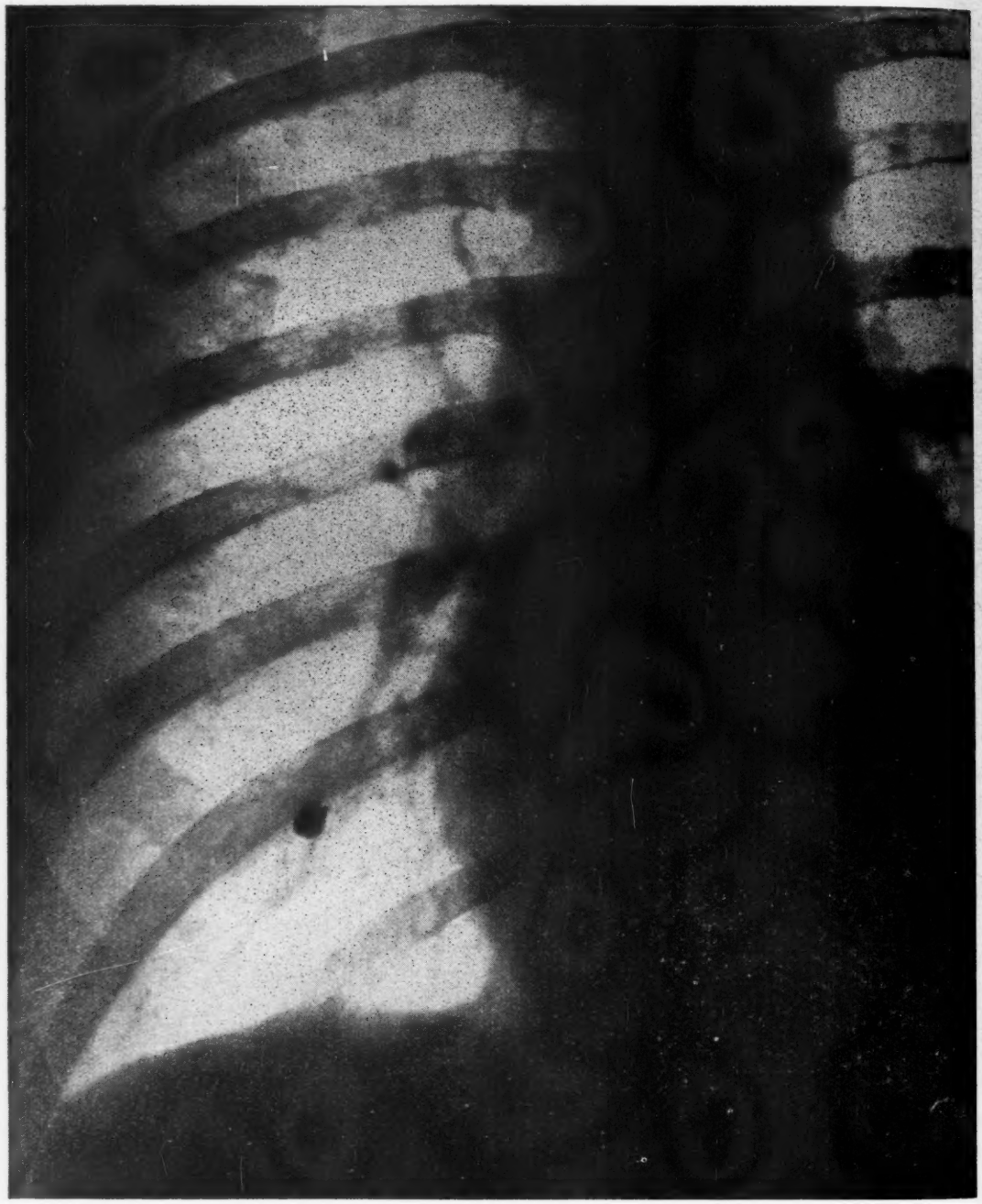
CASE V

Showing temperature chart during patient's stay in Hospital.



CASE VI

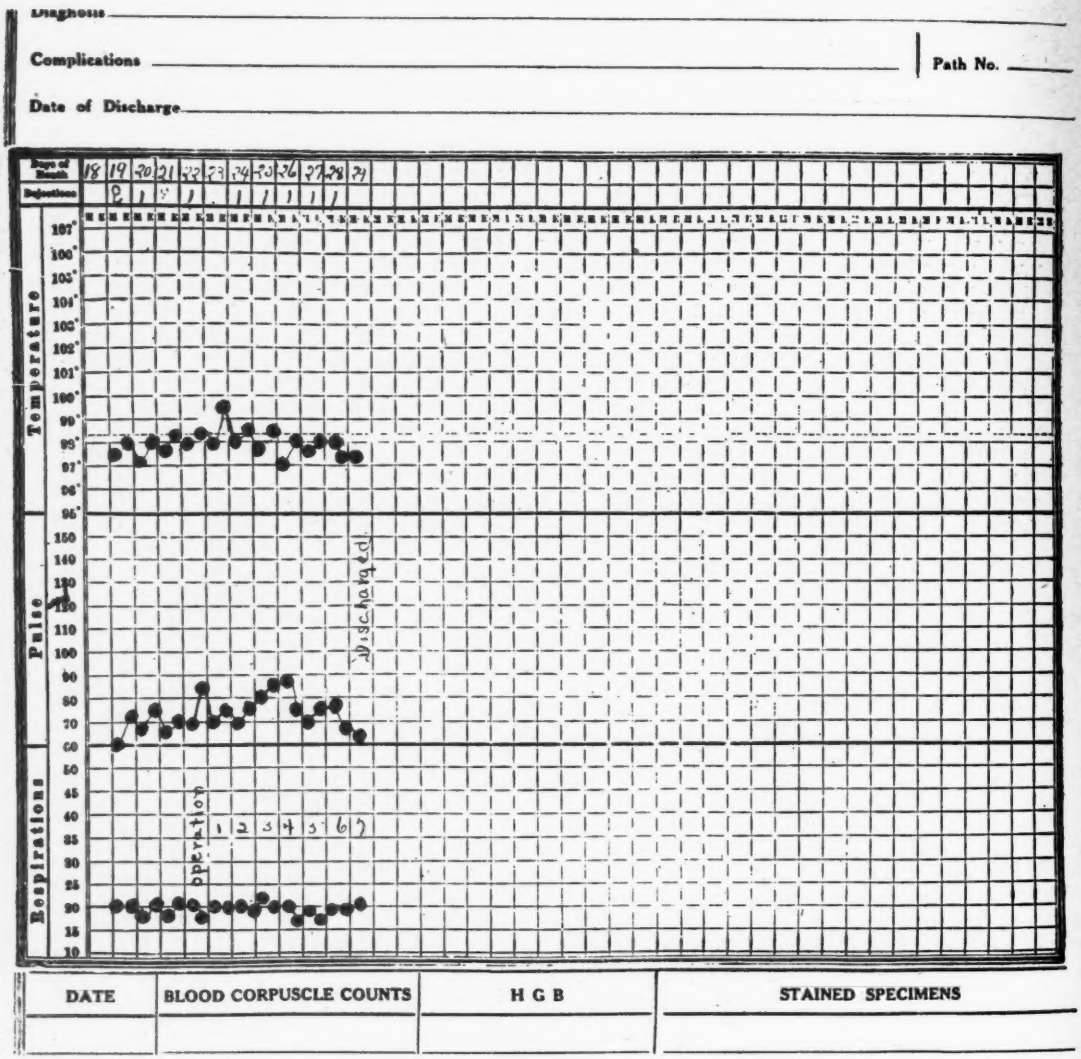
Showing ordinary carpet tack in the right lung for 3 weeks. An attempt was made to remove it under fluoroscopic control. At the glottis it was lost and immediately coughed by the fluoroscopist in the left bronchus, from which it was easily and quickly removed.



CASE VII

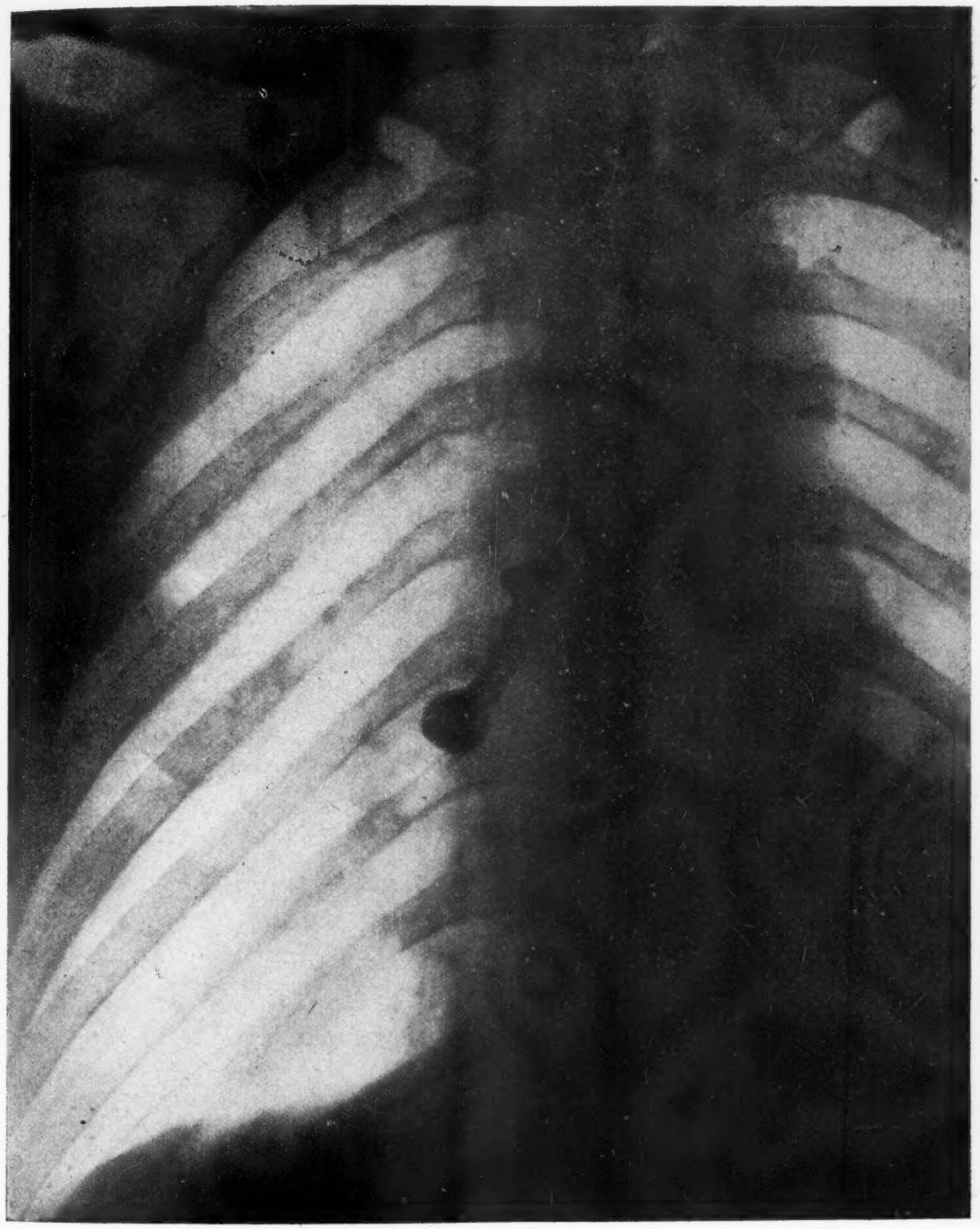
Showing the gold head of a scarf pin in the right terminal bronchus. This foreign body had been lodged in this location for over 30 years. After locating the terminal bronchus, which was stenosed, and dilating it, the foreign body was located and removed under fluoroscopic control.

CASE VII
Lateral view of same patient.



CASE VII

Chart of the same patient while in the Hospital. Note that the removal of foreign body in this patient required 45 minutes, the patient being under general anesthesia. Notwithstanding this, there was no reaction following the operation



CASE VIII

Showing a bridge of teeth in the right bronchus which had lodged there for 2 months. The removal of this foreign body was guided by fluoroscopic control.

without any. The X-ray at this time revealed what was left of the scarf pin, namely, the head, in a dorsal terminal bronchus. By roentgenogram and fluoroscopy the shaft appeared to be gone.

The history of this case together with the preoperative study by roentgenogram and fluoroscopy made me quite sure that there was considerable atresia of the mouth of the affected bronchus.

It was therefore determined to make no attempt by direct vision, so I immediately resorted to the fluoroscope. When the bronchoscope was passed down to the bottom of the main bronchus, the pathological terminal bronchus was easily recognized by its very small lumen and the red areola about it. Following dilatation of the opening there was a gush of mucous tinged with blood. It was impossible to feel the foreign body either with forceps or probe, although told by the fluoroscopist that I was up against it. From this point onward I was entirely directed in my movements by the fluoroscopist and finally succeeded in dislodging it from the anterior wall of the bronchus. Although the operation lasted about forty-five minutes there was no after reaction followirg it, as shown by the temperature charts.

This patient was operated on under general anesthesia and it was very pleasing to me that an assistant was able to hold the foreign body stationary by controlling the movement of the diaphragm on the right.

CASE VIII. J. N. Aet. 40.

Bridge of teeth in right lung inhaled two months ago during an attack of "uremia". X-ray showed a large tooth bridge at the bottom of the right bronchus.

Under a local anesthesia a nine mm. bronchoscope was passed and the foreign body easily recognized, with some granulations about. This foreign body was grasped by direct vision but extracted under fluoroscopic control. When tension was started to draw the foreign body away, considerable was required, and in the fluoroscope the bronchus was seen to come with it. Suddenly the foreign body was dislodged from its bed and came so easily that the operator thought he had lost it, but was immediately informed by the fluoroscopist what had happened and that he still had a firm grasp of the foreign body, which was easily removed.

CONCLUSIONS

(1) Fluoroscopy should be regarded as a first aid and not as a last resort in the removal of opaque foreign bodies from the bronchial tree.

(2) It should be at hand in all cases where the foreign body has been in the lung for a long period, making the chances of seeing it by direct vision or of feeling it, very small.

(3) It should be used after one attempt at removal by direct vision has failed, provided the attempt has been made by a competent bronchoscopist, and everything possible has been done at the first trial.

(4) The mortality is not appreciably raised by this method.

(5) The anesthetic should be selected according to the conditions to combat.

(6) Fluoroscopic removal is preferable to removal of the foreign body by tactile sensation alone.

(7) Under general anesthesia the excursion of the foreign body can be controlled by an assistant fixing the diaphragm on the side of the intruder. This is of very great assistance in accurate grasping of the intruder. This point, so far as I know, is new.

(8) Provided no acute condition is present, the duration of bronchoscopy in adults is of secondary importance, the most important thing being gentleness, which means lack of any trauma. In other words, the bronchoscopist should not strive primarily for speed, but should rather exercise the greatest patience and his manipulations should be the most gentle possible. This conclusion is not based on the series of cases reported in this paper alone but rather the result of my observations in quite a considerable number of foreign bodies in the air passages of fairly long duration.

(9) An expert fluoroscopist is necessary and should share fifty-fifty any success achieved by the operator.

MASSACHUSETTS TUBERCULOSIS LEAGUE

THE FUTURE RESPONSIBILITY OF RESEARCH IN TUBERCULOSIS*

BY WM. CHARLES WHITE, M.D.†

"NATURE cures all curable diseases". This was the opening sentence of the professor of pharmacology in the school of medicine in which I took my education. He did not appreciate the significance of his words for he thereupon began in a long series of lectures to list the evidences to prove that man is the sole agent of nature and recorded the empiricism of the past and present to guide the class before him. He was however but repeating what Paracelsus in 1538 in his *Labarynthus Medicorum* had stated in these words—Let it be known then to all men that had not God created and placed in the bodies of men natural remedies and a natural physician then notwithstanding all the efforts of our physicians not a single creature of earth would remain alive.

I quote them but to emphasize that we have not advanced in tuberculosis beyond nature's cure in spite of all the funds and effort we have spent on its study. For all our vaunted organized attack over a quarter of a century almost all we can offer to the sick is the rest in bed to allow the body, if it can, to accomplish a cure. We have learned means of enhancing this but daily with all the care that can be exercised many slip to the great beyond; a total of deaths that makes us hesitate in our conceit.

What doth it profit a man to say—Tuberculosis is curable—when man does not control the cure. I doubt if any physician in the whole history of the world can raise his hand and say "I cured this case": yet through all the ages the majority of those suffering from tuberculosis have been cured by nature.

Let me show you some of the questions of tuberculosis with which we are faced over the short series of years in which we have reliable records. We are prone to think that the world began and ends with us but as a matter of simple fact it stretches from infinity to infinity. We have few accurate records up to one hundred years ago but such as we have constitute our only guide to the future.

We have just passed through an early diagnosis campaign. I am afraid of the word "early" which we use so carelessly in this regard. "Early" refers to time but we have forced upon it another meaning before the public without thought of the impression such a meaning may give. For every physician who deals with tuberculosis "early" really means a small amount, and we know that many cases diagnosed with the greatest skill at the earliest possible moment go on to death sometimes with great rapidity in spite of every care. But we go merrily forward with an educational campaign that assures the lay mind that if he but come *early* his salvation is sure. We are creating therefore in the public mind a thought from which we shall reap a harvest of mistrust later.

I have another complaint to offer. For fifteen years I lived with advanced and dying consumptives until the question "Doctor, can't you do something for me" forced me to ask myself if by more careful study something could not be done for these doomed cases. A month ago with Dr. Pollak of New Jersey I went through the wards of his excellent sanatorium and again my heart sank within me as I saw the sad eyes of the dying consumptive with the same plaint. It is the cry of the doomed of all time. I think that annually those who conduct the campaign against tuberculosis should visit with a doctor of human sympathy the wards of the dying consumptive so that our thoughts may be guided by increasing effort toward that knowledge which will enable us to stop the ravages of this disease.

There is a group of other facts that have come forward in the last decade that also force us to pause in our enthusiasm of education based on insufficient knowledge.

1st. The war period and the rise in the mortality curve during the years the war continued. This was a shock to many who felt secure in a declining death rate which however is known to have long preceded the campaign efforts.

2nd. The shift in the mortality rate for young girls to the left. It will scarcely profit us if our potential mothers die before the maternity period.

*Remarks made before the Annual Meeting of the Massachusetts Tuberculosis League, Springfield, Mass., April 30, 1928.

†For record and address of author see "This Week's Issue," page 920.

3rd. The rise in tuberculosis in pigs contrary to our predicted results which were based on the decline of tuberculosis in cattle.

4th. The rise in tuberculosis in chickens as a regional and spreading malady of fowls.

5th. The decline of mortality of tuberculosis in the colored race keeping pace with the decline in the white race. And yet for the colored race we can scarcely claim a beginning of our control methods.

The National Tuberculosis Association appreciating these perplexing questions with a foresight characteristic of its work from the beginning appointed a Committee on Medical Research early in 1915 with the entrusted duty of systematically searching for more accurate knowledge of this universal acid fast group of parasites in its relation to disease of man and animals.

It is a pleasure to me to come to Massachusetts at the request of Dr. Kendall Emerson, your President, to discuss the work of this committee because this whole country in its early history owes much to this state. If one reads the record of the early days of Massachusetts and the United States it is quite clear that many of our present methods began here. The first Board of Health, the first State Sanatorium, the first evidence of the relation of soil and moisture to tuberculosis, the first evidence of the relation of pure water to the declining death rate from tuberculosis and other maladies,—all began here. Whether Massachusetts is still in the forefront of the good health movement I leave you to answer.

Names stand out in the early history of this state and the United States that are mileposts in our national public health history. Bowditch, Derby, Sedgwick, McNutt, Smith and many others. The story of Henry I. Bowditch, the father of a former president of this society, Dr. Vincent Bowditch, and his observations on soil and moistures are especially interesting today as we are now considering a study of the fate of tubercle bacilli in the soil concerning which we know nothing and which returns in principle to these early studies and observations of Dr. Bowditch. Then one is constantly brought back to the studies of Segdwick and McNutt on the influence of cleanliness in the shape of pure drinking water and proper sewage disposal on the decline of many of our infectious diseases including tuberculosis. Therefore I repeat that I am glad to be here at Dr. Emerson's request as he is one of the men such as Pope speaks of whose sympathy rises from self till it includes all mankind as the spreading wave of a disturbance in the surface of a placid lake.

A responsibility for expenditure in research you will realize was a serious one and yet it seemed a sure duty of the National Association and its state units and no one cared to shirk it. A small committee appointed worked for two years and then came forward with a tentative plan which the association adopted and work was slowly begun.

This plan contemplated:

1st. A chemical fractionation of a chosen tubercle bacillus strain on synthetic medium and a biological study of the influence of each fraction isolated on the normal and tuberculous animal.

2nd. A comparison with this base line organism of all the different strains of tubercle or acid fast bacilli.

3rd. A study of the living chemistry of the cells of the body in which the bacillus grows.

4th. A study of the problems of light waves in connection with these two living cells which grow together and cause our greatest economic disease problem.

Many fundamental problems were carefully planned and charted before beginning work for, as Schiller said, "The head must plan . . . before the hand can execute". You will appreciate the supreme nature of this task when you see the extent of its organization. The National Tuberculosis Association of which, under the guidance of Dr. Emerson and Mr. Kiernan, you are one of the great units, has been successful in obtaining the coöperation of the U. S. Public Health Service, the Bureau of Animal Industry, the Rockefeller Institute, eight universities and two large drug manufacturers who are all banded together for the purpose of getting at the fundamental laws which govern the development of this disease in man and animals. Already after seven years of coöperation a complete revolution in thought has occurred and six of our large states and cities have joined the coöperation by making gifts to help this endeavor—Chicago, Pennsylvania, Philadelphia, Maryland, District of Columbia, Beaver County and the Rockefeller Institute. They have acted thus generously and unasked and solely because their leaders recognize the importance of the search for new knowledge by newer and better methods developed in the primary fields of research. All of their money for these grants, save in the case of the Rockefeller Institute, was raised by the Christmas Seal. Of the 5% which the states send to the National Tuberculosis Association thousands of dollars are spent in this way although the actual amount spent by the association is small in comparison with the actual expenditure; for, from each grant made we receive as a result the guidance and help of the best research men in the United States; all overhead cost such as building, equipment, light and heat; all of which is saved our annual budget.

When an economic question of such importance to human welfare as tuberculosis is the call all men unite their efforts to secure its solution. It is with a reverent honor for mankind that I record the generosity of our best observ-

ers in these fields in this endeavor to find the fundamental laws underlying tuberculous infection.

I can do no more than touch upon some phases of this work. Just as in nature the most powerful factors are of a comparative rarity and hence their value, so in the tubercle bacillus apparently its most potent elements are there in minute amounts. For this reason in chemical analysis, to secure a sufficient quantity for later biological study one must start with enormous masses of the living organism. A mass of bacilli about the size of an American cheese is the unit of the first steps. To secure this the manufacturing houses, such as Mulford Company and Parke, Davis & Co., must be called into coöperation. During the past three years H. K. Mulford Co. have expended at least $15,000 in the production of the bacteria for our studies at no cost to the association. From the manufacturing houses the material goes to Yale and Chicago for fractionation and from there to the Rockefeller Institute, Cornell Medical College and Vanderbilt University for biological study.

The base line for further studies is the human tubercle bacillus against which will be compared the avian tubercle bacillus, the bovine tubercle bacillus,, the lepra bacillus and various other strains known to have specific characteristics.

From all of the planned and charted studies it is hoped, and now with a certain assured success, to reconstruct the fundamental chemistry of these tubercle organisms and their host cells in the body; and finally to arrive at the means of controlling and curing this sickness.

Massachusetts should certainly, as a premier state, be a part of this endeavor.*

*Dr. White's paper was illustrated to make the points clearer.

NEW ENGLAND SURGICAL SOCIETY

ENTEROSTOMY IN OBSTRUCTIONS AND PERITONITIS*†

BY HOWARD M. CLUTE, M.D., F.A.C.S.‡

THERE are two well established indications for making an enterostomy in some portion of the intestinal tract. These are (1) for the treatment of intestinal obstruction and (2) for the introduction of food, fluid or drugs into the intestinal canal. Recent experience indicates that enterostomy is of some value also in the treatment of peritonitis.

Theories as to the actual agent which causes the symptoms of marked toxemia and collapse so commonly seen in acute intestinal obstruction are numerous but the toxic product that produces the symptoms has not yet been isolated. The material containing the toxin must be in the contents of the intestine, however, which are found proximal to the point of obstruction. Hausler & Foster[1] have shown that the nature of the toxin is more serious when there is, in addition to a simple obstruction, some injury to the intestinal walls such as strangulation. The toxic product, however, whether arising from a simple obstruction or from a strangulated obstruction is to be found in the intestinal lumen just above the point of obstruction. Drainage of these toxic products is the primary reason why enterostomy has been proposed in acute intestinal obstruction and, in my opinion, is the chief indication for the use of enterostomy in intestinal obstruction.

The changes in blood chemistry that accompany acute intestinal obstruction are now well known—the rising non protein nitrogen and carbon dioxide combining power of the blood and the reduction in blood chlorides. It is also generally admitted that many of the symptoms of acute obstruction can be alleviated, some very toxic cases returned to health and convalescence from operation made much smoother by the giving of sterile normal salt solution in large amounts and thus returning the blood chlorides to a normal level.

While attention to the changes in blood chemistry that accompany acute obstruction, particularly the administration of salt solution is of very great importance, the drainage of the toxic products of the obstruction from the intestine must be carried out in most cases if the patient is to recover. The drainage of these products that have formed in the gut as a result of obstruction may be obtained in either one of two ways. The first is by normal bowel movements after relief of the obstruction. The second is by enterostomy at some point above the point of obstruction.

Enterostomy as a method of treating intestinal obstruction has been in use for at least 25 years. Hübener[2] reports that Professor Heidenhain recommended its use at the 31st German Surgical Congress in 1902 and that he reported 5 cases with 4 recoveries. Victor Bonney[3] recommended jejunostomy in peritonitis in 1910 and in 1916 reported 6 cases with recovery.

Since this time numerous surgeons in this country and abroad have recommended the use of enterostomy alone or in conjunction with measures to relieve the obstruction in patients having acute intestinal obstruction with or without peritonitis. Its most general use, however, has been in intestinal obstruction.

It is extremely difficult to analyze the results of enterostomy in obstruction. We must so constantly compare actual results with the results that we believe might have been if our procedure had been different. All surgeons have doubtless operated upon a very sick case of long standing intestinal obstruction, relieved the obstruction, performed an enterostomy and seen the patient recover, and they have seen exactly similar cases recover equally well with relief of obstruction and no enterostomy. Yet they believe, and in my opinion it is true, that enterostomy is of value in intestinal obstruction. It is logical to assume that the drainage of highly toxic material from the gut in these cases is beneficial. In statistics dealing with the value of enterostomy in obstruction we include cases that are moribund or nearly moribund on whom we do an enterostomy as the one possible chance of saving the patient's life. Death in such a case should not be held against enterostomy but rather against delay in surgical treatment.

In a study of a large series of cases of intestinal obstruction Van Beuren & Smith[4] find that the statistics do not prove that enterostomy has lowered the mortality rate. They admit that enterostomy has been used in only a small percentage of cases (32%) of acute ileus and that it is too often reserved for the exceptionally poor risk cases. These statements, however ,do not alter the very strong belief held by most surgeons that enterostomy should be done in acute obstruction as an important part of the mechanical treatment of the case. Doubtless it is not done often enough.

We have performed an enterostomy on 44 patients having intestinal obstruction. Of these 27 or 61% recovered and 14 or 31% died. Three cases could not be traced.

Seventeen of these 44 patients had obstruc-

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tion in the large intestine for which a tube was sutured either in the cecum or in some more distal portion of the large gut. 13 of the 17 recovered, 2 died and 2 were not followed. Of the two who died, one developed pneumonia some days after operation. The other patient died of peritonitis which arose from leakage around the tube that was sutured into a greatly distended sigmoid colon.

It is evident that when obstruction occurs at the lower end of the intestinal tract that cecostomy can usually be depended upon as a life saving measure and should be used. It is also evident that the greatest care must be taken to prevent leakage about the drainage tube since one death occurred from this cause in this group of enterostomies.

In 21 patients, enterostomy was done at some point in the ileum usually a short distance above the point of obstruction. 9 of these patients recovered and 11 died. One patient was not traced. These figures are surprising. It had been thought that ileostomy done above a low obstruction would in theory at least give the best chance of success. These 21 cases include both primary and secondary ileostomies and of course in many instances the ileostomy was done as a measure of last resort.

In six cases with intestinal obstruction, a jejunostomy was done with 5 recoveries and one death. All of these jejunostomies were done for obstruction coming on from 1 day to 1 week after operation. In no case was any procedure undertaken to relieve the actual obstruction and all save one recovered.

It is well known that the higher the obstruction the more rapid and more serious is the course of acute obstruction. This fact has been repeatedly demonstrated both experimentally and by clinical observation. In this connection it is important to note the recent experimental studies made by Haden & Orr[5] with high jejunostomies in dogs.

They found that dogs with high jejunal drainage lived only 2 to 5 days, a shorter time than when simple obstruction was caused at the same point, and they found that with a high jejunostomy chemical changes occurred in the blood similar to those of high intestinal obstruction. They showed, however, that an ileostomy was compatible with life. These facts are worthy of careful note when we consider the point at which we shall drain the intestine in acute obstruction and they have a definite clinical application in the treatment of drainage from the intestine once it has been established. A jejunostomy, we believe, should be allowed to drain for but 36 to 48 hours after it has been made since longer drainage may seriously deplete the body of fluid and chlorides. After this the tube should be clamped off. At four hour intervals the tube should be flushed with normal salt solution and three or four ounces of this left in the jejunum at the close of the irrigation. Glucose solution in addition to the salt solution may also be given through the tube. If free drainage occurs through the tube it should not be continued for a longer period (36 to 48 hours) than seems necessary to remove the toxic products of the obstruction.

We have seen no case in which we felt any harm had resulted to the patient from jejunal drainage. All our enterostomy tubes are inserted by either the Witzel method or with a double purse string suture. Whenever possible omentum is interposed between the intestine and the abdominal wall. In no case has there been a persistent intestinal fistula after the enterostomy tube has been removed. Most of the tubes come out on the 6th or 7th day after operation with immediate closure of the fistula.

It is but reasonable to assume that since a paralysis of the intestine accompanies peritonitis, that enterostomy should be advocated in the treatment of this condition. Indeed it is highly probable that some of the cases of so-called obstruction that were not relieved by enterostomy were in reality cases of peritonitis with paralytic ileus. Thus Heidenhain in his original suggestion for the use of enterostomy spoke of its value in the obstruction seen in peritonitis. So also did Victor Bonney and some of the cases he treated were cases of obstruction with peritonitis. Wilkie[6] states that a post mortem study of cases dying of so called peritonitis 5 to 10 days after operation for acute appendicitis showed that intestinal obstruction accounted for the death of 75 per cent.

It is true that as peritonitis advances, paralysis of the musculature of the involved loops of bowel becomes more prominent. At first this paralysis is largely localized about the source of the peritonitis but as the infection spreads a generalized distention of the gut occurs. This comes from increasing paralysis of sections of the gut and from distention of the remaining gut with gas and fluid proximal to the paralyzed portions. There is in peritonitis not only a toxemia from the bacterial infection of a very large endothelial lined cavity but also an acute intestinal obstruction.

Certain degrees of peritoneal infection can be withstood and overcome by the human organism. It is conceivable that the deciding factor in the recovery from peritonitis is in the degree of intestinal obstruction that occurs with it. It is well known that the differentiation of acute intestinal obstruction from acute diffuse peritonitis is very difficult and at times impossible. The clinical picture of each condition and also the changes in the blood chemistry are at times identical.

It is therefore logical to assume that treatment which is of value in acute intestinal obstruction will be of some benefit in peritonitis. In peritonitis we drain the peritoneal cavity in order to reverse the direction of lymph flow and bring antibodies to the infected area. The lumen of

TOTALS

	Cases	Dead	Recovered	Unknown
Peritonitis				
Jejunostomy	7	3 (1 embolus, 1 pneumonia, 1 peritonitis)	4	
Ileostomy	6	5	1	
Cecostomy	8	5	1	2 (probably died)
Total in Peritonitis	21	13	6	2
		61%	28%	
Obstruction				
Jejunostomy	6	1	5	
Ileostomy	21	11	9	1
Cecostomy	17	2	13	2
Total in Obstruction	44	14	27	3
		31%	61%	
Ulcerative Colitis	1		1	
Feeding	8	7	1	
	74	34	35	5
		Dead	Recovered	Unknown

the intestine should also be drained of the products of the obstruction that have accumulated above the paralized portion of the intestine. Lavage of the intestine in certain cases will remove the toxic contents whose presence would otherwise be the fatal factor in the combination of peritonitis and acute intestinal obstruction.

Our experience with enterostomy in peritonitis has been limited to 21 cases in which peritonitis was obviously present. It is doubtless true, however, that in some of the 44 patients on whom we report enterostomy for obstruction, there was also present some degree of peritonitis. The 21 cases of frank peritonitis were treated with the usual methods for dealing with peritonitis and in addition had an enterostomy performed. 6 recovered, 13 died and 2 were not traced but probably died.

It is of interest to consider these cases more particularly. In 8 of the 21 patients a cecostomy was done because of the marked distention of the large intestine. Only 1 recovered, 5 died and 2 cases, occurring very early in our experience are of unproven outcome as the result of incomplete records, but may be assumed to have died.

Although the number of cases is small it seems reasonable to state that cecostomy is not of any special advantage in peritonitis.

In six cases of peritonitis an ileostomy was done. 5 died and only 1 recovered. Here again we may rightly infer that ileostomy is not of remarkable value in peritonitis.

In 7 cases a jejunostomy was performed in addition to the usual measures carried out in the treatment of peritonitis. Of these 4 recovered, 1 died of embolus several days after operation, 1 died of pneumonia 5 days after operation and 1 died of peritonitis. It seems fair to say that jejunostomy plus other treatment cured 6 out of 7 patients with peritonitis.

From this small group of 21 cases in whom the intestine has been drained for peritonitis we are struck with the relatively greater benefit which has been derived when the drainage has been instigated high in the intestinal canal. And if we consider that in draining the intestine in peritonitis we are attempting to treat simply the factor of intestinal obstruction which accompanies the peritonitis, the results seem consistent. Some years ago it was our practice to drain the *dilated* intestine at the point of obstruction. It now seems more advisable to go above the point of marked dilatation and paralysis and drain the intestine which is not involved in the paralytic process. Here the gut has not yet lost its power of contraction and many of the toxic products arising from the obstruction can be obtained from the tube.

The writer has operated recently upon two patients with perforating appendicitis and diffuse peritonitis by performing an appendectomy with drainage and then doing a jejunostomy through a separate upper left abdominal incision. In each case purulent material with gross signs of peritonitis filled the pelvis and also escaped from the incision made in the left upper quadrant. Each patient made a surprisingly complete and uneventful recovery. We quite realize that it is possible that each patient might have recovered without the jejunostomy but in our judgment this is very unlikely.

These and other similar experiences lead us to

believe that a jejunostomy should be added to the usual surgical treatment of all cases of diffuse or generalized peritonitis.

CONCLUSIONS

1. Enterostomy in acute intestinal obstruction is of value when it removes the toxic products collected above the point of obstruction.
2. Drainage of the large intestine in low obstruction gave excellent results. Drainage of the jejunum in acute obstruction gave good results.
3. Septic peritonitis produces death largely from toxemia arising (1) from the infection and (2) from the products of the intestinal obstruction caused by paralysis of the gut.
4. Cecostomy in peritonitis gave very poor results; ileostomy in peritonitis gave very poor results; jejunostomy in peritonitis gave quite satisfactory results.
5. Jejunostomy should be added to our present methods for the treatment of diffuse septic peritonitis.

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DISCUSSION

DR. R. C. COCHRANE, Boston: I think you will all agree that this is a very important subject and that this is a very interesting paper on this subject. I agree thoroughly with what Dr. Clute had to say about the value of enterostomy in obstruction. I think we have all gradually more and more come to doing a high enterostomy rather than draining the intestine close to the obstruction, and in my experience jejunostomy has worked very well in cases of obstruction.

I would like to lay a little stress on the use of hypertonic salt solution and the use of glucose in these cases. The ordinary normal salt solution is of great value, and fluids must be used, but in certain cases which didn't yield to any other treatment we have found that the use of a small amount, 250 c.c., of a hypertonic salt solution intravenously gave relief of symptoms where the ordinary salt solution wasn't effective.

It has also been my experience in these post operative obstructions where you do a jejunostomy as a life saving measure that you don't have to do another operation to relieve the obstruction. We found that the obstruction relieved itself spontaneously, and the bowels moved spontaneously in a week or ten days. If a subsequent operation becomes necessary, it should be done early and not later than a week or ten days, otherwise if the obstruction still persists, the patient will die of starvation. This isn't so important in enterostomy lower down.

When it comes to the subject of peritonitis, I don't think I can wholly agree with Dr. Clute. From my experience at the City Hospital in Boston I haven't seen any value from enterostomy or jejunostomy in any real peritonitis cases. You are dealing there with a paralytic ileus with coils of intestine distended with toxic material, and I don't believe you can get rid of that with a jejunostomy. I think you are going to have coils of intestine that you can't drain. Of course, most of the cases of post operative obstruction are due to a more or less localized peritonitis, just how extensive it is I don't think any of us can tell exactly; and that is why statistics on these cases do not necessarily prove the point. In a great many of these cases of obstruction you can't tell when you do them whether they are due to an actual obstructing band or a localized peritonitis or a generalized peritonitis. You can't tell until it is too late.

So it is justifiable to go ahead and do your enterostomy before you make your diagnosis; but it has been my experience that the cases that got well were cases of obstruction rather than cases of peritonitis.

DR. IRVING J. WALKER, Boston: I quite agree with Drs. Clute and Cochrane as to the value of jejunostomy in obstruction of the small intestine, due to mechanical factors.

My experience with jejunostomy for the relief of ileus secondary to general peritonitis has been disappointing. I wish to speak briefly of thirty-eight cases of the mechanical type of obstruction which followed from five to twelve days after operation. All of these were treated by jejunostomy, with a mortality of 25.6%.

For the most part, the mortality was due to delay in doing the enterostomy, or to the fact that the obstruction was complicated by an attending or subsequent general peritonitis. Of those that recovered, 70% were relieved by no other operative measures than jejunostomy.

Where jejunostomy does not relieve the obstruction, and where there is an existing septic wound, or an infection within the peritoneal cavity, you have a most unfortunate situation, since exploration for the relief of the obstruction itself is quite likely to result in a mortality from general peritonitis.

Dr. Cochrane spoke of operating for removal of the obstructing element where the bowels do not move through the natural passage following jejunostomy. Where this becomes necessary, operation should be done within the first week following jejunostomy. After that time, and in spite of glucose and chlorides being given, the patient will be found to have approached a considerable degree of starvation and hence will be a poor risk for operation.

I wish to stress the value of the blood study in these cases of obstruction. Laboratory work has repeatedly demonstrated that with the con-

dition of obstruction there is always a high nonprotein nitrogen, a loss of chlorides, and hypoglycemia. With these conditions one must give salt solution and glucose before and after jejunostomy and until the blood chemistry has approached normal.

I want to mention one other use of jejunostomy for those who are interested in stomach surgery. In extensive stomach operations where resections were called for and where food could not be given by mouth for four or five days, I have recently been doing jejunostomy in addition to the stomach operation. Making use of the jejunostomy opening has made it possible to introduce nourishment at a much earlier time than would be otherwise possible.

DR. C. C. LUND, Boston: It is very kind of you to allow me to speak, and I want to ask one question of Dr. Clute on the question of peritonitis—some of the patients dying of peritonitis apparently die largely of dilatation of the stomach. Now, of course, a jejunostomy will probably prevent that. However, when I was a resident on Dr. Richardson's service we used an in-lying stomach tube with great success in many cases. Of course, it stops the vomiting at once, and I wonder whether it wouldn't accomplish as much as the jejunostomy. I understand the use of such a tube passed through the nose has become a routine in the Children's Hospital in Boston with splendid results in cases of peritonitis. I haven't had cases of that kind and haven't had the opportunity of trying it in children, but if it works in children I think it might work in adults.

DR. H. M. CLUTE, Boston (closing): I don't think we can prove certainly from our experience that enterostomy saves patients with peritonitis. As I said before, it is extremely difficult to analyze the results. So much is based on one's impressions. You do an enterostomy, the patient recovers and then you compare that fact of recovery with the mental idea you had beforehand that recovery was impossible.

I quite agree with Dr. Cochrane and Dr. Walker that in many cases of peritonitis jejunostomy won't be a bit of use and I don't anticipate that it will save every patient. You can only try it, but I believe firmly from what I have seen that some of these patients on whom we did jejunostomy and enterostomy who by all the rules of previous experience should have died recovered with the jejunostomy. If only a few out a hundred do that, it is worth while because I don't believe we have shortened the period of life of the fatal cases by doing the jejunostomy.

I have been interested in what Dr. Cochrane and Dr. Walker said about hypertonic salt solution. Dr. Cochrane told me they used 250 c.c. of five per cent. salt solution intravenously and got better results from that than from normal salt solution. We haven't tried that but have tried using normal salt solution in large amounts. Doubtless it would be of advantage to use some hypertonic salt solution but we feel there is much value in the larger amounts of fluid given when normal salt solution is used.

We have had no experience with the in-lying duodenal tube. We have tried passing a stomach tube through the nose, two tubes, one for irrigation and one for drainage but we haven't had much success and doubtless it may have been due to our inability to keep the tube down properly. I think that in theory it is reasonable and doubtless it should be tried more frequently.

I can't say anything more about peritonitis than that if I find a patient has peritonitis, I shall do a high jejunostomy in the belief that I will do no harm and with the hope that the removal of the toxic products of obstruction from the upper bowel will be the deciding factor in the patient's recovery.

FIBROLIPOMA, REPORT OF A CASE

BY JOSEPH S. BARR, M.D.*

THIS case is reported, not because of the rarity of the condition, for lipoma is perhaps the most common of benign tumors, but because of its unusual size and accessibility.

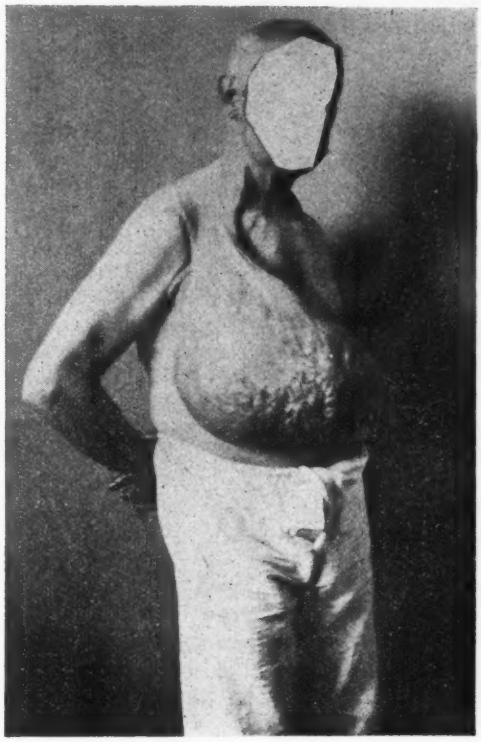
G. W., Surg. 29908, entered the Peter Bent Brigham Hospital Oct. 20, 1927. He is a married electrician, age 58. The family and past histories are irrelevant.

Eight years ago he first noticed a small growth, "like a wart", in the region of his right clavicle. It has increased gradually but constantly in size until the present time. For the past several years he has had to have his clothes altered to accommodate the tumor beneath them. It occasions him no pain or discomfort except for a slight sense of dragging weight on the shoulder. It has not interfered with his work; the day before entry he was on a step-ladder cleaning windows. He seeks relief on the advice of the company physician, who discovered the tumor in the course of a routine examination.

Physical examination is essentially negative except for the local examination which reveals a pedunculated tumor the size of a foot-ball, depending from the right supraclavicular region. The pedicle is 3.5 cm in diameter and contains numerous blood vessels, over which a distinct bruit is heard on auscultation. The tumor itself is 36 cm long and 28 cm in diameter. The skin is intact over the growth, but there is marked hyperkeratosis with deep fissures over the lower part of the mass. It is completely anaesthetic below the pedicle. There is no regional lymphadenopathy. X-ray examination of the lung fields is negative. The laboratory findings are essentially normal except for a moderate quantity of sugar in the urine.

Oct. 26, under gas-oxygen anaesthesia, the tumor was amputated. The pedicle was found to be very vascular. All blood vessels were ligated and the skin closed without drainage. The postoperative course was uneventful and the patient was discharged Nov. 3, with the wound healed.

The pathological report stated that the tumor weighed 7 kilos. The diagnosis on the microscopic section was "Lipoma with fibrosis and elephantiasis of the skin". The specimen has been placed in the Warren Anatomical Museum.



*For record and address of author see "This Week's Issue," page 920.

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 14171

FATAL CHOREA

MEDICAL DEPARTMENT

A sixteen-year-old schoolgirl entered March 13.

Five weeks before admission she had a streptococcus sore throat. She had been ailing since that time. Three weeks before admission she had "rheumatism" in her feet, ankles, hip and wrist. March 5 her face began to twitch. March 8 she stopped school. The following day she talked incoherently. March 10 she began to throw her head from side to side. The night of March 11 she was found in her night clothes crouching over the register talking to herself about being chilly. The bed was pulled to pieces and the room torn up generally. The day before admission she began to throw herself about and was unable to talk at all. She had a wild night in spite of chloral. She had not slept for five or six nights.

She had a similar attack at twelve years.

Clinical examination showed a well nourished girl showing marked incoördinate movements of the entire body, requiring forcible restraint to keep her in bed. She did not speak when questioned. Cheeks and mucous membranes flushed. All prominences and convex surfaces of the body showed reddening in the form of discrete and confluent papules, not decolorizing on pressure. Skin dry and harsh. Apex impulse of the heart felt in the fifth space 8 centimeters from midsternum. Action rapid, regular. Sounds of fair quality. No murmurs. Pulmonic second sound accentuated at the base. Lungs normal. Abdomen held rigid, otherwise normal. No edema. Pupils not examined. Knee-jerks, abdominal reflexes and reflexes at wrist and elbow not obtained. Plantar reflexes normal. No Kernig. Neck not stiff.

Amount of urine not recorded, specific gravity 1.022, slightly alkaline at both of two examinations, cloudy at one, a very slight trace to the slightest possible trace of albumin at both, acetone at one. Blood 19,000 to 14,000 leukocytes, polymorphonuclear leukocytosis, hemoglobin 85 per cent.

The patient was very violent. Her movements were only partly controlled by chloral and occasional doses of morphia. March 18 a short rough high pitched systolic murmur appeared at the apex.

March 19 a lumbar puncture was done and 15 cubic centimeters of fluid withdrawn, part of it very bloody, part nearly clear. It reduced Fehling's solution slightly. Sediment from the clearer tube was mostly blood corpuscles. Of 100 cells 80 per cent. were polymorphonuclears, 20 per cent., mononuclears. The number of these cells seemed to be no more than might be expected with the amount of blood present. A culture of the fluid showed no growth.

Temperature 99.9° to 103.7°, with a terminal rise to 108°. Pulse 108 to 160. Respirations 15 to 28.

After the lumbar puncture the temperature rose steadily to 108°. The patient became weaker, the movements subsiding only as her strength failed. Strophanthin was given with no response. A double parotitis developed. The breathing became very shallow. The morning of March 20 the patient died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

I remember this patient well. She was in my service seventeen years ago. Of course the diagnosis is perfectly obvious—chorea—and it is very tragic when we see a case as terrible as this. When one has been fishing and gets a fish it flaps around in the bottom of the boat. She flapped around like that, and only tremendous doses of drugs had any effect. She was terribly violent.

NOTES ON THE PHYSICAL EXAMINATION

Of course we were watching the heart, knowing there must be something there in such a case as this, as there practically always is. For five days we undoubtedly were looking as often as we could under the difficulties that one can imagine in examining a patient who was in such motion.

I do not know why we did that lumbar puncture. I do not see to-day any sufficient reason for it. The case does not resemble meningitis or any other cerebral disease that could involve the meninges. Part of the fluid was bloody, undoubtedly because we got some blood mixed in with the tap. We got no information. We had a sediment showing the blood introduced during the tap and no evidence of a meningitis. The negative culture was of some value.

We do not always realize the fevers of acute chorea, although in this case, if I remember rightly, there were some other reasons besides the chorea.

DIFFERENTIAL DIAGNOSIS

Remembering the case as clearly as I do during life, I do not think any diagnosis was or should have been seriously considered except

the so-called chorea major, the severest form of chorea. We have had three fatal cases of this disease in this hospital to my knowledge during thirty years. Two of them, as I remember, showed at necropsy something more besides the chorea. Whether this is one of them I cannot remember.

In chorea the lesions in the heart at necropsy are almost constant, and they are usually of fairly uniform type,—little spots of soft reddish material as big as the end of a pin, arranged in rows along the edge of a valve, usually the mitral; never, so far as I know, large vegetations, and never any chronic or ulcerative process unless there has been something more besides chorea. That is, that endocarditis, as I remember it, seems to me different from the average endocarditis of rheumatic heart disease or of bacterial endocarditis.

Two earlier English observers, Poynton and Paine, who wrote a good deal on rheumatic infections, described brain changes in chorea. So far as I know those have not been backed up by later observations. I do not believe there were any found here. There must be changes in the brain, and why we do not find them I do not know. The symptoms are all brain symptoms. This is strikingly evident in hemichorea, one half of the body perfectly quiet and the other half, including the face, twitching. That could not be anything but a brain lesion.

Another point not always referred to is the mental changes in chorea, which get covered up because we suppose that the patient's queer sounds and queer remarks are due to the mechanical disturbance of speech. But there are also definite mental changes, as one would expect in a brain disease such as chorea certainly is.

What is the relation of chorea to acute articular rheumatism? I am confident that they are the same disease with different manifestations. Just as we can have tuberculosis either in the joints or in the meninges, so we can have the organism of rheumatism, never yet discovered, either in the joints or the brain of such patients as we are studying today. One gets every possible sequence and combination of rheumatism and chorea: one sees first rheumatism and then chorea, one sees first chorea and then rheumatism, one sees the two at once.

The hyperpyrexia here is of some interest. The hyperpyrexia of rheumatism is referred to in all the classical accounts of acute articular rheumatism. Here it followed a lumbar puncture which may have had something to do with producing it. But my guess is that the lumbar puncture did not produce it. I believe that the asepsis even at that period was tolerably good. It is not so easy to introduce sepsis into the cord or it would happen oftener. So that I doubt whether anything will be found in the cord, if it was examined, to account for that temperature of 108°. I guess it is a hyperpyrexia of chorea parallel to the hyperpyrexia of acute rheumatism.

I do not know anything about the finding of the Aschoff bodies. Do you find those in chorea, Dr. Mallory?

DR. TRACY B. MALLORY: I think we probably find them in the heart. I do not know of any reports of them in the brain.

DR. CABOT: If they do occur in the heart there is no reason why we should not find them here. I should say the post-mortem ought to show small vegetations on the mitral valve, possibly on other valves, and nothing else that we can predict.

A PHYSICIAN: Is there any significance in the parotitis?

DR. CABOT: I do not know how to say anything about that. I do not remember it in chorea cases before. But any patient who is gravely sick is apt to develop a parotitis owing to the spread of mouth organisms up Steno's duct. As we do not know the origin of parotitis we shall not be able to find out whether this is a choreic parotitis or some other kind, I suppose.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Chorea major.
Acute endocarditis.
Exhaustion.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chorea major.
Acute endocarditis.
Parotitis.

ANATOMIC DIAGNOSES

1. *Primary fatal lesions*

(Acute chorea.)
Verrucose endocarditis of the aortic and mitral valves.

2. *Secondary or terminal lesions.*

Hemorrhagic areas of the lungs.
Slight bronchopneumonia.
Fatty metamorphosis of the liver.
Slight cystitis.
Parotitis.

DR. MALLORY: The necropsy findings were not extensive, as the head and cord were not examined. The heart showed, as predicted, minute vegetations on the mitral and also a few on the aortic valve. There was no deformity of any of the valves. The lungs showed a very slight acute bronchopneumonia, and also numerous petechial hemorrhages that could not be attributed to a pneumonic process. The kidneys showed a slight degree of tubular degeneration but no glomerulonephritis.

Bacterial cultures were made of heart's blood, both aerobic and anaerobic, and no organisms were grown.

DR. CABOT: Was the brain examined?

DR. MALLORY: No.

It is interesting to hear Dr. Cabot mention again Poynton and Paine, because the present trend of work on rheumatic fever seems to be swinging back decidedly towards the point of view of some of the early workers. There seems to be no question that in a very considerable proportion of cases of rheumatic fever it is possible during life or post mortem to grow a streptococcus from the blood stream. The figures from various laboratories differ a great deal, but there are a number of laboratories in which fifty per cent. of cases of severe rheumatic fever seem to show this organism. We are still quite a way from proving that that is the cause of rheumatic fever, but some of the recent work is at least suggestive, particularly that of Small of Philadelphia and Birkhaug of Rochester. They have grown streptococci which produce neither hemolysis nor methemoglobin. Birkhaug has shown that this organism produces a relatively mild exotoxin of about one-fourth the strength of scarlet fever or erysipelas, and that patients with rheumatic histories tend to react with positive skin tests to this toxin, whereas normal controls are generally negative. He found that he himself was a susceptible individual as measured by the skin test, and injected a small quantity of this toxin into one of his joints, thereby producing clinically a very typical attack of acute rheumatism, not only that joint but several other joints swelling, becoming red and very painful, with fever, the condition lasting about four days. Injection of the organism in rabbits produces mitral endocarditis, but only when huge quantities are given. So far the work stands about there, but it does seem possible that we may come back to some of the opinions held by the earlier investigators.

DR. CABOT: I am very glad to hear your opinion of that work of Dr. Small's. I had rather thought that most of the laboratories were not backing that up.

DR. MALLORY: I am inclined to pin more faith on the others. Birkhaug's work was more carefully controlled and his claims are less sweeping. The organisms have been worked with here in Boston and also a similar organism isolated by Clawson and Bell of Minneapolis. All three of these organisms are apparently serologically closely related. One can also, it is true, recover it from the throats of a very large number of normal persons.

DR. CABOT: But that is nothing against it, do you think?

DR. MALLORY: Not at all.

CASE 14172

AGE AS A FACTOR IN PROSTATECTOMY

UROLOGICAL DEPARTMENT

An American of eighty-two entered through the Emergency Ward November 7 complaining of urinary frequency, nycturia, and difficulty in starting the stream.

He had had these symptoms three years. The stream had gradually decreased in power. Two years ago all the symptoms increased in severity until he had acute retention, requiring catheterization twice a day for twelve days. Operation was discussed but not done because of his age. He improved, but the symptoms continued, growing slightly more severe. He passed small amounts of urine every half hour. For the past year he had had dizziness, dyspnea and precordial pain on exertion, and orthopnea. He had rare epigastric distress. In the past few days the dizziness had increased until he could not stand for any length of time, and frequency had increased so that he had slept little at night and had had urgency and pain by day, relieved by the passage of even small amounts.

In the Out-Patient Department the day of admission he had a residual of sixteen ounces of cloudy urine. A note from a physician of the hospital staff stated that he had eighteen ounces residual November 6.

A number 18 catheter passed easily. The prostate was large and smooth.

No family history or past history is recorded.

Clinical examination showed a well preserved old man appearing years younger than he was, in no obvious discomfort. Heart enlarged to the left. Sounds of poor quality. Rhythm very irregular, at times absolutely. Rate slow but occasionally increased. A loud harsh blowing systolic murmur throughout the chest, best heard at the apex, transmitted to the aortic area. A loud high pitched rough sound localized to the aortic area. Blood pressure 110/70. The sphygmomanometer shut out many of the sounds at the wrist. Occasional strong beats came through down to 70. An electrocardiogram showed auricular premature beats, at times in runs, rate 90 to 130, marked left axis deviation. Abdomen large, full, pendulous. Bilateral direct inguinal herniae. Hemorrhoidal tabs. Prostate symmetrically enlarged, firm, elastic, not tender. Slight edema of the right ankle. Varicosities of both lower legs. Pupils and knee-jerks normal.

Before operation amount of urine 48 to 98 ounces, urine turbid, alkaline, specific gravity 1.019, a slight trace of albumin, occasional red blood corpuscles. Blood not recorded. Wassermann negative. Renal function November 9: appearance time 18 minutes, in two hours 50 per cent.; November 16 appearance time 6½ minutes, in two hours 53 per cent. Non-protein nitrogen 35 milligrams.

Before operation temperature normal except for one rise to 100.7° the day before operation, pulse 60 to 100, respirations normal except for one increase to 31 November 12.

X-ray showed the transverse measurements of the heart increased. The heart shadow was prominent in the region of the left ventricle.

The supracardiac shadow was wide, prominent both to the right and to the left in the region of the aortic knob. In the oblique view it was not definitely dilated.

A heart consultant reported: "... No definite story of angina. Marked left ventricular enlargement, rough aortic systolic murmur and thrill, absent aortic second sound, blowing apical systolic murmur, plateau pulse. Blood pressure 150/90. Irritable heart with premature beats, not heard today, November 14. In this case I should suggest pre-operative digitalization. He is a fair ether risk. Lung bases clear."

The patient was put upon constant drainage. He remained in the same condition. November 17 operation was done. He was not in poor condition after it. The temperature was flat. The fluid intake however was found very low, so that a subpectoral had to be given November 18. That evening the blood pressure fell to 72. He was given intravenous glucose and put on shock blocks. He came back very well and continued to do well until November 21. Then he began to take less fluid and was perhaps mildly delirious at night. He continued to lose strength. November 24 there was some hemorrhage from the wound and the blood pressure was reported as 72, but by the time the house-officer arrived it was 115. November 26 he was worse, flushed, breathing with difficulty and rather drowsy. The lungs showed moist râles. There was edema over the shins. The night of November 26 the temperature was 103.7°, the pulse 152, the respirations 32. November 27 he died.

Discussion

BY EDWARD L. YOUNG, JR., M.D.

At his age, eighty-two, a man complaining of frequency, difficulty in starting the stream and urination at night means prostatic obstruction in the majority of cases. Occasionally in old men we see such symptoms coming from hypertension and arteriosclerotic nephritis. As a rule stricture will make itself known before this. Another thing of interest is that these symptoms were present for only three years. Symptoms of prostatic obstruction as a rule begin earlier than at seventy-nine. It is a disease which most commonly makes its first appearance in the neighborhood of sixty or sixty-five, although owing to the slow progress of the disease the patient may not present himself to a doctor with it for a number of years after that. It always brings up the suspicion of malignancy when the symptoms appear and come to a crisis in a relatively short time. It by no means proves malignancy, but it always arouses that suspicion.

Two years ago, when eighty, he had acute retention. The account of the events following that of course rules out the question of stricture. If he had been having acute retention from stricture he would not get by with that casual announcement that twice a day he required catheterization for twelve days. There is one criticism that I want to bring out in regard to the treatment. When any patient has to be catheterized because of acute retention, in order to have that treatment efficacious it has to occur oftener than twice in twenty-four hours. Either the patient has to be on constant drainage or else the bladder must be emptied at least three and sometimes four to six times a day during the first of the time at least.

Age alone is not a criterion for refusing operation, assuming that the condition is a benign adenomatous prostate. Of course it is true that the older the patient the greater is the likelihood that his urinary tract and his cardiovascular system are so damaged that he will not stand operation. But the basis for operation should be the condition particularly of those two systems, taken in connection with that indefinite something which explains the general resistance of the individual as the surgeon sees him.

He refused operation here, and because of that refusal has gone on having more and more trouble. When a man with obstructing prostate reaches the stage of acute retention one of two things ought to be done. Either he must be kept on a catheter life until we know that he is taking care of himself adequately, or else he must be operated on. So that again I think my second criticism is that they let him go on causing himself more and more damage.

Eighteen ounces residual means a good deal of back pressure damage to the kidneys. The fact that it was infected adds still more danger to the kidneys.

50 per cent. is a very good renal function for a man who has had as much damage as that. The good renal and non-protein nitrogen tests back each other up.

So far then the heart is the bad feature of the picture. They are doing everything they can to find out the condition of the two main systems to see whether they will stand operation or not.

Operation was done ten days after he came in. I do not know just what they are going to do, whether they will do a two-stage suprapubic operation or a one-stage perineal. As a rule I think the routine operation of least risk is a two-stage suprapubic operation. The first stage of the opening of the bladder can be done under local anesthesia with practically no shock to the patient. It results in several things. First, it gives better drainage of the urinary tract. Second, it gives a chance for the suprapubic wound to get stabilized if there is infection, so that in the second operation there is no shock or spreading of sepsis in putting the finger into the original incision. He has had drainage all the time. I am assuming that they did a two-stage operation. It is true that a perineal operation done under spinal anesthesia gives a very low mortality. But there is in a perineal operation a greater danger of poor urinary control after operation than in the suprapubic approach.

X-RAY INTERPRETATION

The heart findings are those of arteriosclerosis.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Obstructing prostate, perhaps malignant.

PRE-OPERATIVE DIAGNOSIS

Obstructing prostate.

OPERATION

Local novocain. Preliminary ligation of the vasa was done. A median suprapubic incision was then made through the skin, subcutaneous tissue and sheath of the rectus. The prevesical fat was identified and the peritoneum reflected. A cystotomy opening was made in the usual fashion. Exploration with the examining finger disclosed a large intravesical prostate with no apparent abnormality of the bladder wall and no calculi.

FURTHER DISCUSSION

If he could not stand that I feel that there is very little chance of ultimate success. A patient who will not stand that operation is in such a condition that his expectancy of life is too slight to be seriously considered anyway. He would be pushed out of the picture with almost anything that came up.

That whole picture seems to spell failure of the cardiovascular system or the cardiorenal system to carry on.

It is often a very hard question to decide in a man of this age whether to keep him on a catheter life or whether he can stand operation. I remember one instance when I was assistant to Dr. Hugh Cabot in 1914. A patient was refused operation partly because of his age, partly because of his general poor condition. He was put on catheter life, and has been on catheter life ever since. He is now in his nineties. He has said several times that he wishes he had taken the chance of operation. Catheter life means very great care for the details of treatment,—catheterization by the clock regardless of the patient's feelings, dependent on the amount of residual, and if necessary an inlying catheter at night. A year and a half ago there was a patient on whom it was absolutely impossible to operate. He was two weeks in the hospital with a non-protein nitrogen over 100 and a zero red test. I told him to go out on catheter life, including an inlying catheter at night, and told him he could come back when he was better. I told his wife he would be dead within three months. He showed up two months ago with a non-protein nitrogen ranging about 40. He had lived literally according to the directions that I gave him, and things having gone smoothly he did not report back. His prostate is now out and he is back at work. So that catheter life can be carried out by the average intelligent person and he can live a comfortable existence, although it is never so satisfactory as a successful operation.

I do not know what the necropsy findings will show, whether they will show death from cardiac failure.

A PHYSICIAN: Could it be a low grade bronchopneumonia?

DR. YOUNG: Perfectly well. It has been ten days, and with a certain amount of kidney decompensation and edema at this stage of the game I do not know why there could not be a certain amount of pneumonia.

I was talking about age and prostatectomy with one of the men yesterday. He told me of a man of ninety-six who had a successful prostatectomy. The oldest person I have personally done is eighty-six. When the man I talked with was an interne in this hospital during the Spanish War this patient entered because of prostate. He was a veteran of the Mexican War in 1847. So that they can be fairly old, assuming that they are good patients to stand the reaction.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Benign hypertrophy of the prostate.
Myocarditis.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Obstructing prostate.

ANATOMIC DIAGNOSES

1. *Primary fatal lesion.*
   Hyperplasia of the prostate.
2. *Secondary or terminal lesions.*
   Pulmonary embolism.
   Chronic cystitis.
   Chronic pyelonephritis.
   Bronchopneumonia.
   Arteriosclerosis.
   Arteriosclerotic heart disease—aortic stenosis.
   Hypertrophy of the heart.

DR. TRACY B. MALLORY: The primary cause of death was, of course, hypertrophy of the prostate. Both lateral lobes were greatly enlarged, each measuring 4 centimeters in diameter. The middle lobe was also enlarged and pedunculated, so as to form a ball valve in the orifice of the urethra. The bladder was dilated, hypertrophied, and showed numerous small diverticula. The ureters, particularly the left, were dilated, and the pelvis of the left kidney was much increased in size. Numerous small abscesses containing colon bacilli were present in the left kidney. The right kidney was negative except for a slight degree of arteriosclerosis.

His failure to withstand the operation probably rests with the condition of his heart. This was much hypertrophied, weighing 555 grams, with a preponderant hypertrophy of the left ventricle. There was a very marked degree of aortic stenosis, confirming the physical signs described by the cardiac consultant. It was of a pure sclerotic type without evidence of rheumatic involvement.

A slight bronchopneumonia was present in the lower lobe of the right lung.

The immediate cause of death was a pulmonary embolus which arose from a thrombosis of the right femoral vein just below the inguinal ligament.

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THE ONE HUNDRED AND FORTY-SEVENTH ANNIVERSARY EXERCISES OF THE MASSACHUSETTS MEDICAL SOCIETY

THE Society accepted the hospitality of The Worcester County Medical Society and the Worcester Chamber of Commerce and held its Annual Meeting in the Bancroft Hotel and Worcester Chamber of Commerce. The previous Annual Meeting held in Worcester was in 1851.

The weather was not propitious, both days being rainy, but with the exception of the opportunities to visit the city at large and enjoy golf nothing interfered with the regular program although it is probable that a considerable number of fellows were kept from attendance by the unpleasant conditions.

The Section Meetings were very well attended, the largest numbers being attracted to the Section on Surgery although there was sustained interest in all of the other exercises and the accommodations were none too ample for the audience. It was the general impression that the selection of the subjects which were discussed reflected great credit on the officers of the sections. The discussions following the leading papers brought out all possible amplifications of coördinate problems. The management of the meetings was excellent and there was little loss of time. The audience in each section paid close attention to all that was said.

The registration showed six hundred and one doctors and seventeen guests which is a creditable number considering the weather.

One hundred and ninety-eight participated in the banquet which was served immediately after the conclusion of the Annual Oration by Dr. Walter B. Cannon. This oration was scientific with certain practical applications of interest to practitioners and appears in this issue.

The speaking program was well arranged, the addresses being both entertaining and instructive.

The Council Meeting was well attended and consumed more time than any previous meeting by this body lasting until after four o'clock with only a short intermission for luncheon.

Many of the subjects provoked animated discussions and several amendments to motions were made before final decisions were reached.

The amended code of by-laws was adopted as submitted by the Committee and endorsed by the Society the next day. This document should be carefully kept for reference for there are several important changes.

The adoption of the minority report submitted by one member of the Committee on Insurance demonstrated the renewed approval of the policy of the Fidelity and Casualty Company.

Although several reports of Committees were subjects of animated debates the President kept the members strictly to the matters on hand and no unnecessary time was devoted to irrelevant subjects.

So much important business was transacted that it is the duty of all who feel any responsibility in the functions of the Society to read the reports of the proceedings which are being prepared by the Secretary and which will be published as soon as possible.

The list of officers elected appear on page II of this issue.

Taken all in all this anniversary meeting can be regarded as one of the most instructive and interesting in the history of the Society. Credit was given to the committees in charge of details by appropriate votes.

THE ANNUAL MIGRATION

THE camp and cottage season is descending once again upon the city dweller, and with it, as usual, certain health hazards are entailed which his physician should warn him against. Established cottage sites and camp locations frequently fail to have adequate sanitary conven-

iences and unimpeachable water supplies, and as always the milk question, particularly when young children are in the family, may not receive sufficient consideration. It is a common belief that, since cows are found most numerously in the country, country milk must on that account be preferable to city milk. It is also a fact that in this present day typhoid fever is rather a disease of the country than of the city, and despite the fact that our neighbor farmer may be an upright citizen and a steady church goer his cows may not share his religious beliefs to the extent of drinking only from controlled water supplies or allowing themselves to be milked only by healthy milkers.

As an unofficial health officer the family physician should instruct his patients to drink only pasteurized milk, and if adequately pasteurized milk is unobtainable, it should be pasteurized or boiled in the home. Sources of drinking water need investigation, and if there is any question whatsoever of the purity of the supply it should be state-tested. Pending a favorable report, or if such a report cannot be secured, the drinking water should be boiled.

For all travelers—and in these days of the ubiquitous motor cars traveling is an almost universal way of vacationing, frequently employing roadside camping—certain immunizing procedures should be carried out; vaccination against smallpox, vaccination against typhoid fever and toxin-antitoxin immunization to diphtheria. It must be remembered that diphtheria immunity may take as long as six months to develop, and a last minute inoculation is of no value.

Smallpox and typhoid fever are slowly on the increase, we are becoming careless in our contacts, and our contacts with all manner of people from all sorts of reactions are rapidly increasing, for never has the general movement of people been so great as in this restless age. The open road is appealing, and close to the bosom of nature is the ideal environment for this vacation, but remember that most places are now visited by man and man too often leaves pollution in his wake.

IS ALUMINUM A DANGER TO HEALTH?

We have been asked for an opinion with respect to the effect on health of the use of aluminum cooking utensils.

It has been claimed by a dentist in Ohio that cancer is caused by eating foods prepared in aluminum utensils.

The question was submitted to Dr. George H. Bigelow, Commissioner of the State Department of Public Health and he in turn referred it to Lawrence T. Fairhall, Assistant Professor of Physiology of the School of Public Health of Harvard University. He reports that investigations in the laboratory of the Lancet in 1913 later confirmed by Doctors John Gaister and Andrew Allison at Glasgow University warranted the conclusion that "the use is not attended with any risk to health of the consumers of food cooked therein" provided that "ordinary precautions" are observed.

This last qualifying phrase leaves the matter open to doubt because the average cook may not know the necessary precautions, but it is generally conceded that the use of strong acids or alkalies in aluminum utensils had better be avoided.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Cannon, W. B. A.M., S.D., LL.D. (Hon.), M.D. Harvard, 1900. George Higginson Professor of Physiology Harvard Medical School, since 1906. His subject is: "The Mechanism of Emotional Disturbance of Bodily Functions". Page 877. Address: Harvard Medical School, Boston.

Levine, Samuel A. A.B., M.D. Harvard, 1914. Instructor in Medicine, Harvard; Associate in Medicine, Peter Bent Brigham Hospital. His subject is: "Some Unproved Impressions Concerning the Subject of Heart Disease". Page 885. Address: 270 Commonwealth Avenue, Boston.

Smyth, D. Campbell. A.B., M.D. Harvard, 1909. Associate Laryngologist, Massachusetts General Hospital; Bronchoscopist, Thoracic Clinic, Massachusetts General Hospital. His subject is: "The Fluoroscopic Removal of Metallic Foreign Bodies in the Bronchi". Page 887. Address: 375 Commonwealth Avenue, Boston.

White, Wm. Charles. M.B., M.D. Toronto Medical School, 1901. In charge Tuberculosis Research, Hygienic Laboratory, Washington, D. C.; Chairman, Committee on Medical Research, National Tuberculosis Association; Director, National Tuberculosis Association; Vice-Chairman, 1927-1928, Div. Med. Science, National Research Council; Chairman, 1928-1929, Div. Med. Sciences, National Research Council; Chief Bur. Tuberculosis, Am. Red Cross (1917-1918) in France, later in Italy. His subject is: "The Future Responsibility of Research in Tuberculosis". Page 905. Address: 2334 19th Street, Washington, D. C.

Clute, Howard M. B.S., M.D. Dartmouth, 1914. F.A.C.S. Assistant Surgeon, New England Deaconess Hospital and the New England Baptist Hospital. His subject is: "Enterostomy in Obstruction and Peritonitis". Page 908. Address: 605 Commonwealth Avenue, Boston.

Barr, Joseph S. S.B., M.D., Harvard, 1926. House Officer, Huntington Memorial Hospital, 1924-1925; Surgical House Officer, Peter Bent

Brigham Hospital, 1926-1927; Orthopedic House Officer, Children's Hospital, 1928. His subject is: "Fibro-Lipoma, Report of a Case". Page 913. Address: N. E. Peabody Home for Crippled Children, Oak Hill, Newton Centre, Mass.

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Serology

The modern uses of sera in the diagnosis and treatment of disease, like many another treatment with which we are familiar, harks back a long way for the inspiration which has resulted in present day enthusiasm for their employment. At the period when the Alexandrian School of Medicine was the conspicuous center of medical education certain Arabian physicians were experimenting by feeding ducks, which had been slowly immunized by the ingestion of materials ordinarily poisonous to them, to human beings that they themselves might be rendered immune against these same toxic substances. Some will doubtless recall the enthusiasm with which Sir Almoth Wright's investigations were received, not so many years ago now, and how some were led to hope that the promise held out by the "opsonic index" was soon to revolutionize not only treatment but the diagnosis of many obscure conditions. Indeed it is perhaps strange that more thought had not been given this subject in view of the fact that it was long ago abundantly well known that spontaneous recovery, uninfluenced by any treatment is constantly taking place and it would seen but natural to suppose that antidotes were being supplied from within the body. With such knowledge as was possessed, for several hundred years, of the circulation and even certain of the properties of the cytologic elements of the blood it would seem that the idea of the Arabian physicians should not have been allowed to lie dormant so long. On the other hand when one dips into the literature pertaining to this subject, as viewed by the modern bio-chemist, he finds himself over his depth before he has gone any distance from the shore. He must learn to think in terms of agglutination, complement fixation, precipitins, antigens and a maze of terminology, which leaves him either hopelessly despondent as to any immediate future for practical serum therapy or wildly enthusiastic to engage in the task of discovering the magic key to these most interesting physiologic problems.

How complicated the interrelation of physiologic processes is, we can glimpse now and then in ways which are so striking that we may only stand aghast at the complexity of the problems to be solved before any clear preception of what is going on within us may be had. We regard hate as a most undesirable quality in man's character and yet there was a time in mankind's history when it doubtless played an important rôle through its power to preserve, through the survival of the fittest, those physical qualities best designed to advance the human species along the road to its ultimate goal. It did this by playing upon certain physiologic stops about which we have only comparatively recently known anything, viz: the ductless glands. Hate and its confrère, anger, by stimulating the adrenals, poured into the circulation that which enabled primitive man to overcome odds that would have been overwhelming to him had he not been possessed of this, today, undesirable quality. Under such stimulus we now know man can exert many times the energy he is capable of without such influence. What this doubtless meant to primitive man in the sudden exigencies of his wild life may be easily imagined. The individual who had this quality in its most unbridled state was destined to be the victor in his many conflicts. Among the higher, dare we say spiritual, attributes of human nature at the dawn of human history, hate and anger occupied an important position, which happily is being surrendered to ones more useful to the present phase of man's evolution. It is however such examples as this that serve to indicate the complexity of the problems that the student of physiologic chemistry must perforce solve. The pathway of any major physiologic process, fundamental to life, is one continuous series of interrelated events each one of which is preparing the way for the next higher step, all of which must be taken in an absolutely orderly fashion and with an immediacy which is unthinkable, as witness the extraordinary rapidity with which the adrenalin reaction is brought about under the excitation of anger. Indeed upon this very immediacy alone does its value to the organism depend. How far we are from the knowledge of these matters which will put us in possession of the means of influencing these processes we cannot know.

It is obvious that the lesson we should learn is that ill-advised experimentation, while not perhaps dangerous, is unscientific and often impracticable and that the sane thing to do is to encourage the investigator and discourage all attempts to exploit undetermined physiologic reactions as panaceas in the way of treatment.

MISCELLANY

TUBERCULOSIS ABSTRACTS

The quest for a cure for consumption is probably as old as history. Koch raised hopes of finding a specific cure in tuberculin, the concentrated media of broth cultures of tubercle bacilli. His failure has since been followed by numerous others. In the files of the National Tuberculosis Association there are today records of 680 "cures" some that give pause to thought, some ingenious, but most of them ridiculous. Meantime, rest, fresh air, and good food have been established as the tripod on which the treatment of tuberculosis depends. Latterly rest has been so unanimously emphasized by clinicians

who specialize in tuberculosis that it might be regarded as the pillar of successful treatment, while fresh air, good food, artificial pneumothorax and other therapeutic devices might be considered as supplementary supports.

REST IS RELIEF FROM STRAIN

Rest may mean the sloth of the indolent or the relief from tension that follows change of occupation, says Allen K. Krause. Therapeutically however, rest represents *relief from strain.* Treatment must aim to limit and confine the activities of tuberculous foci and to reduce to zero or a minimum the absorption of harmful focal products. At any time, undue stress may stir quiescent foci into renewed activity. It is axiomatic that uncontrolled movement of a diseased or injured part will promote the spread of the disease and delay recovery. To

Of all the countless remedies proposed, rest alone has stood the test of time.—*Gerald B. Webb.*

stop the progress of tuberculous foci is to cure tuberculosis.

Fever, fatigue, loss of appetite and other constitutional symptoms of tuberculosis are manifestations of intoxication resulting from absorption of focal substances. The rate and capacity of this absorption depend on the circulatory and respiratory activities of the body. Rest brings about a diminution of physiological demands and reduces the amount of focal absorption.

Rest for the sick man is a better "tonic" than exercise. As a result of prolonged rest, the appetite returns, the fever falls and a sense of well-being sets in, while depleted reserves are built up, thus assisting in the healing of foci. Rest is a potent medicine, to be prescribed according to the requirements of each individual case by a physician who understands its use.

The febrile, acutely ill cases must have absolute bed rest for at least two weeks after the temperature has returned to normal. After the constitutional symptoms have disappeared, the patient must still be kept below the fatigue line. The fatigue line is an individual affair, registered only in the patient's own consciousness. The duty of the physician is to explain to the patient why relief from strain is important. But there can be no set formula for the individual patient; he must rely on his own intelligence and behavior. Rest should be so engraved on the patient's mind that he will automatically respond with rest to the first symptom of fatigue.

Sanatorium treatment is vastly more satisfactory for the majority of patients since rest and discipline and the means of insuring these are more readily obtainable there. The sanatorium, moreover, teaches and trains the patient how to care for himself.—*Rest and Other Things, Allen K. Krause, Williams and Wilkins Company.*

FOOD REQUIREMENTS AND FRESH AIR

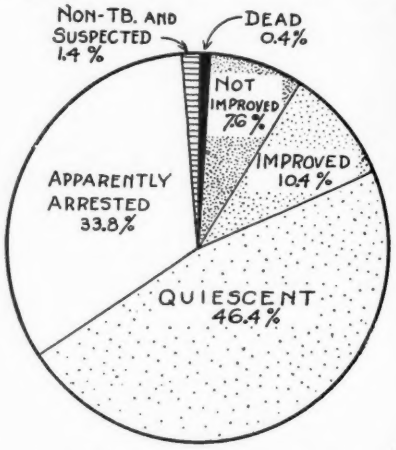
Good nutrition is important, but "stuffing" the patient, as formerly practiced, is a mistake. Overeating is like clogging an engine with unburnt carbon by using too much fuel. Sometimes, the appetite must be cajoled. Three good meals a day, two or three glasses of milk (with or between meals), one or two eggs a day, are often sufficient to add enough to the patient's weight to bring him the gain wished for. A good general rule is that the least amount of food that will enable any patient who is underweight to gain up to and slightly beyond the normal weight is the optimum diet for that patient.

Fresh air as a "cure" for tuberculosis has probably been overemphasized by the laity. It is, however, an essential aid to recovery. Outdoor air is a mild and beneficial stimulant. Sleeping out of doors does not necessarily hasten recovery, provided eight to ten hours a day are spent in the open air and the night passed in a well-ventilated room. Mere dryness of the air is of little avail. Temperature, humidity and air movement determine the quality of indoor ventilation.—*Rules for Recovery from Tuberculosis, Lawrason Brown, Lea & Febiger.*

DISPOSITION OF PATIENTS

Patients may be divided into three groups as far as treatment is concerned:

1. Suspects, cases under observation, and those in which the diagnosis is not definite, can be treated



Condition on discharge of 222 patients who remained over 90 days (average 229 days) at Trudeau Sanatorium, 1925-26.—*Annual (1926) Medical Report of the Trudeau Sanatorium.*

at home or fall into groups (2) or (3). The patient is on trial and more radical measures, such as going to a sanatorium, may be, and very likely will be, necessary. In a few cases of this group, sanatorium or hospital treatment, if it can be obtained at once, is of great value educationally and otherwise and entirely justified in instances where adequate home treatment is not possible in order to clear up a diagnosis.

2. Cases in which the diagnosis is definite and in which the disease is progressive, with or without

a positive sputum, should be sent to a sanatorium or hospital at once and should remain as long as the physician considers it necessary. This is the ideal to be sought for in the great majority of cases. Home treatment may be substituted (a) when there are no children in the family who might be exposed to the disease in the open form, (b) when the intelligence of the patient or his family is such that adequate carrying out of details is assured, (c) when good nursing and medical service is available and (d) when there are facilities for proper outdoor treatment.

3. Arrested, apparently arrested and quiescent cases need close medical and nursing supervision if the good done at a sanatorium is to be permanent. Home treatment may be satisfactory for the majority of these cases. Frequent visits to the home by the nurse and monthly consultations should be required. The amount of work done and the choice of employment are to be decided by the physician. The patient should know that it may become necessary at any time for him to return to the sanatorium when indications of an impending breakdown occur. —*Diagnostic Standards Pulmonary and Glandular Tuberculosis of the National Tuberculosis Association, Seventh Edition, November, 1926.*

CLIMATE AND ALTITUDE

There is no specific for the cure of tuberculosis. Climate is not a specific. Altitude is not a specific. . . . No physician, therefore, is justified in advising a change of climate unless he knows that the patient's financial status will enable him to command the essentials. To put it categorically, if a little arbitrarily: proper medical supervision, sanatorium regime, either in a sanatorium or in the home, reasonable contentment of mind and intelligent coöperation count ninety or ninety-five per cent. of effective therapeusis; climate and change of environment count five or at the utmost ten per cent. Why, therefore, sacrifice the ninety or ninety-five per cent. for a five or ten per cent. in those cases who can *not* command the one hundred per cent.? On the other hand, if the patient can afford to go to a first class sanatorium or secure the services of a good phthisiologist in a more salubrious climate, and will be reasonably contented away from home, by all means he should be urged to avail himself of the full one hundred per cent. of these efficacious measures.—*Louis C. Boisliniere, Journal of the Outdoor Life, February, 1928.*

A CIRCULAR OF INFORMATION

CLINICAL CONGRESS OF THE AMERICAN COLLEGE OF SURGEONS

The surgeons of Boston are planning a highly attractive program of clinics and demonstrations for the eighteenth annual Clinical Congress in that city October 8th to 12th. All branches of surgery will be represented therein, providing a complete showing of the clinical activities of that great medical center. Clinics will begin at 2 o'clock on Monday afternoon and continue throughout the mornings and afternoons of the following days.

Among the distinguished visitors from abroad who will attend this year's Congress and present papers are: Professor Vittorio Putti, Professor of Orthopedic Surgery at the University of Bologna, Italy, and Director of the Rizzoli Institute; Sir William I. de Courcy Wheeler, of Dublin, Past President of the Royal College of Surgeons of Ireland.

The Presidential Meeting on Monday evening will be held in Symphony Hall, as will also the annual Convocation on Friday evening. On the other evenings meetings will be held in the ballroom of the Copley-Plaza Hotel. The annual Hospital Conference opens on Monday morning in the ballroom of the Copley-Plaza with a program of papers, round table conferences, and practical demonstrations dealing with the many problems related to hospital efficiency.

Boston now has ample first-class hotel accommodations for all who wish to attend. In recent years a number of new hotels have been built, including the Statler with 1300 guest rooms. Reduced railway fares from all points in the United States and Canada will be granted for this meeting.

Registration in advance will be necessary as attendance will be limited to a number that can be comfortably accommodated at the clinics. Clinical Congress headquarters will be established at the Statler, where the ballroom and foyer and other large rooms on the mezzanine floor have been reserved for our exclusive use. The ballroom at the Copley-Plaza will be utilized for the evening meetings and other large gatherings.

FRANKLIN H. MARTIN, *Director-General.*

CAUSE OF DENTAL CARIES

Recently, according to the United States Bureau of Education, an experiment was made in a London hospital on the effect of different diets on the teeth. One group of children of about 7 years of age was given daily one and a half to two pints of milk, egg, one and a half ounces of sugar, butter, rice, meat, fruit, bread, vegetables and cod liver oil. Another group had only a half to three-quarters of a pint of milk, very little egg, three ounces of sugar, more potato but less of other vegetables, a little oatmeal, more bread, but no butter and no cod liver oil. At the end of eight months there was an increase of more than twice as many decayed teeth in the second than in the first group, and the extension of caries existing at the beginning was more than two and one-half times as great.

Such an experiment is interesting, but it seems a little like taking liberties with other people's teeth.

NEW YORK ACADEMY GETS DR. STREETER'S COLLECTION

A collection of medical books containing many of the earliest printed works on the subject, brought together by Dr. E. C. Streeter of Boston over a period of twenty years, has been purchased by the New York Academy of Medicine from Dr. A. S. W. Rosenbach, a recent report states.

The purchase price was $185,000, of which $85,000 was contributed by the Rockefeller Foundation.

When our own Academy of Medicine becomes a reality it is to be hoped that New York's gains will no longer be our losses.

RECENT DEATHS

LOTHROP—DR. HOWARD AUGUSTUS LOTHROP, surgeon, died at the Charlesgate Hospital, Cambridge, of sepsis, June 4, 1928, at the age of 63. While removing a dressing he scratched his hand with a safety pin, resulting in infection of his right arm.

It was necessary to amputate the arm, but he was so profoundly septic that he did not survive.

Dr. Lothrop was born in Sharon on December 31, 1864, the son of Horace A. and Sarah Gorham (Swain) Lothrop. He came to Boston and attended the Boston Latin School. In 1887 he received his A.B. degree at Harvard, *magna cum laude*, and he was graduated from the Harvard Medical School in 1891, taking also the degree of A.M. at that time. He studied also in Europe for two years. He served as a house officer at the Massachusetts General Hospital and had practised in Boston. He became associated with the Boston City Hospital as a visiting surgeon soon after graduation. He was instructor in surgery at Harvard Medical School 1903-1912, assistant professor of surgery 1912-1922 and acting professor of clinical surgery 1922-1923.

He was a member of the Massachusetts Medical Society, American Medical Association, the American Surgical Association, Boston Society of Medical Sciences, Boston Surgical Club and the Boston Society of Medical Improvement. He was a Fellow of the College of Surgeons. He belonged to the St. Botolph, the Harvard and University clubs.

Dr. Lothrop, who was unmarried, made his home with his mother at 101 Beacon street, besides whom his survivors include two brothers, John Howland Lothrop of Portland, Ore., and Dr. Oliver A. Lothrop of Boston, as well as three sisters.

COURTNEY—Dr. Joseph William Courtney, neurologist, died at his home in Boston, June 6, 1928, after a short illness with pneumonia, aged 60. He was a graduate of Harvard College in the class of 1890 and of Harvard Medical School in 1893.

From 1893-1909 he was a member of the staff of the Boston City Hospital and for some years physician-in-chief of the department of nervous diseases at the Carney Hospital and the Boston City Hospital. He was a member of the American Neurological Association, Massachusetts Medical Society, and the Boston Society of Psychiatry and Neurology. He was also a corresponding member of the Societé de Neurologie de Paris.

Dr. Courtney was the author of the book "The Conquest of Nerves," and for many years a constant contributor to medical journals. He was a member of Harvard Club and Papyrus and Medical History clubs of Boston, and the Authors' Club of London.

He is survived by his widow and three children, Mrs. John T. J. Clunie of 230 Commonwealth Avenue, Boston; Paul G. Courtney of Weston, and Gerald Courtney of Phoenix, Ariz.

CORRESPONDENCE

SHALL THE INDIVIDUAL PAY THE COST OF ANTI-RABIES TREATMENT?

18 Dalton Road, Belmont, Mass.,
June 5, 1928.

Editor, The New England Journal of Medicine:

The editorial entitled "Rabies" which appeared in the May 31st issue of the New England Journal of Medicine contains this sentence: "If the individual is able to pay, obviously he should pay any costs in connection therewith." The reference is to the cost of the prophylactic treatment and its administration in cases of dog-bite by rabid or possibly rabid animals. The justice of such a procedure is not at all obvious to me. It appears to me to be obviously unfair to ask the individual bitten to pay the cost of the prophylactic treatment when he is not the owner of the dog.

I am presuming of course that it is admitted that rabies is, among domestic animals, primarily a disease of dogs, that human beings are, in most cases, more valuable than dogs, and that dogs, however useful and likable under certain circumstances, are not, for the most part, a real necessity but rather a minor luxury.

We will suppose that, whereas I do not see fit to enjoy this particular luxury, my neighbor or anyone in a locality I may have occasion to visit or which is within striking distance for his dog of my locality, does own a dog. The dog develops rabies. Having the several misfortunes in varying degree of having been born, of having parents who chose to dwell in that particular city or town and of having inadvertently failed to don his bite-proof underwear on the day that he happened on the same street with the rabid dog, my child is bitten. Because I am able to pay and am fool enough to admit it, I am asked to defray the necessary costs that my child may enjoy the experience of from fourteen to twenty-one daily hypodermic injections with all the immediate discomfort and possible more remote sequellae that may go therewith. It is a most naive view of the matter to say the least and the only thing about it which appears to me to be quite obvious is its ludicrousness.

It is possible that there may occasionally be a dog owner who would, under such circumstances, voluntarily offer to defray the cost, but I imagine that such individuals are for the most part living under forced confinement where such luxuries as owning a dog are not permitted. It would also be possible, no doubt, to collect the cost through legal procedure under circumstances where all the necessary technicalities had been complied with and I suppose lawyers must live, too, but the cost of collection might conceivably exceed the amount to be collected, not to mention the effect of such a procedure upon the gaiety of neighborhoods.

Regardless of the amount involved and the ability of the victim or his family to defray the cost, it would seem only fair to expect and require the owner of the rabid dog to pay for any and all treatment required as the result of injury inflicted by such an animal. Where the owner is unquestionably unable to pay or where such injury is inflicted by a stray animal which has been permitted to lend local color to the community through the laxness of the public officials or public opinion, it would seem equally as obvious to expect the city or town to defray the entire cost of treatment.

Such a gross misplacement of the financial burden, great or small, as referred to, not only penalizes the victim, but also relieves the owner of a very proper responsibility for the ownership of a dog. The fact that damages can, I believe, be collected legally for such injury, would seem to prove the existence of such responsibility and serve to substantiate the justice of such an attitude. Moreover, in the case of a disease which is practically 100 per cent. fatal it should not be necessary to await its clinical recognition in order to be able to obtain the cost of prophylactic treatment, no matter how trivial the initial injury.

Yours very truly,

Edward A. Lane, M.D.

INSURANCE FOR PHYSICIANS ENGAGED IN PUBLIC HEALTH WORK

Editor, NEW ENGLAND JOURNAL OF MEDICINE:

At the suggestion of Dr. Safford, I am sending you the enclosed correspondence, with the request that it be published in the JOURNAL.

Sincerely yours,
FRANCIS GEO. CURTIS, M.D.,
Sec'y, Comm. on Public Health.

May 10, 1928.

George H. Crosbie, Esq.,
79 Milk Street, Boston, Mass.

My dear Mr. Crosbie:

At a meeting of the Public Health Com. of the Mass. Medical Society, the question arose whether members of the Society engaged in Public Health work are protected by any of the plans for insurance for members of the Society.

These men are not engaged in general practice, confining their activities entirely to public health.

They are of two classes: those who actually perform certain medical acts, such as vaccination, administration of antitoxin, inoculation against certain diseases, etc., and those who do not actually perform such duties but order their medical employees to do them. These last might be held liable on the theory that the chief is responsible for the acts of his agents.

I was requested to ask you if, in your opinion, these men are covered by any of the insurance plans now in effect and, if not, whether some plan could be arranged whereby such men could receive the benefits of insurance.

Very truly yours,
FRANCIS GEO. CURTIS, M.D.,
Sec'y, Comm. on Public Health.

May 14, 1928.

Francis G. Curtis, M.D.,
Sec., Comm. on Public Health,
City Hall, West Newton, Mass.

Dear Doctor:

Answering your letter of the 10th let me state that we already have a number of the Public Health Physicians insured. They all take the lower rate, that is $20.00 for a $10,000/$30,000 policy, unless they specialize in X-ray work, or in major surgery, in which case they take an increased rate.

I am enclosing herewith a sample policy, with rate sheet for the various specialties, but the major part of these men belong in Group 1. They should all be insured, as there is a chance of suit in which the City will not protect them.

If there is any further information I can give you please let me know.

Yours very truly,
GEO. H. CROSBIE.

NEWS ITEMS

HELPFULNESS OF THE "PROBLEM-CHILD" CLINIC—The Vermont Children's Aid Society in its latest annual report testifies to the helpfulness of the problem-child clinic recently established in the University of Vermont by the National Committee for Mental Hygiene. A staff conference was held each month in connection with the clinic for the benefit of workers of the society and other organizations.—*Bulletin Children's Bureau.*

A GIFT TO THE MELROSE HOSPITAL—Mr. and Mrs. Alfred H. Colby have given one hundred and twenty-five thousand dollars to provide for a new wing for the Melrose Hospital.

DR. BENJAMIN PARVEY VISITS EUROPE—Dr. B. Parvey of Charlotte Street, Dorchester, sailed for Europe on the S. S. Dresden, May 31, 1928, from New York Pier. He was given a send-off by 300 friends at the pier. His absence will cover a period of eighteen months. He will practice and study obstetrics and gynecology at the leading hospitals in Vienna and Berlin. His family will join him in June, 1929, and after touring the Continent for six months he will return to the States.

REORGANIZATION OF THE MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE—The Massachusetts Society for Social Hygiene has been reorganized with Dr. Cecil R. Drinker, Assistant Dean of the Harvard School of Public Health, as President, Dr. George H. Bigelow, Massachusetts Commissioner of Public Health, as Honorary Vice-President, and Dr. Helen I. Doherty McGillicuddy as Executive Secretary.

The Society plans:

1. To continue educational work in social hygiene and venereal disease.
2. To make an intensive investigation and campaign against venereal disease in a single community in the State rather than a diffusion of effort through many of our cities.
3. To coöperate with and further the work of the State-aided venereal disease clinics.

NOTICES

ANNUAL NARCOTIC REGISTRATION RETURNS MUST BE FILED WITH THE COLLECTOR BY JULY 1 OR PENALTY ATTACHES

The forms to be used in applying for reregistry under the Harrison Narcotic Law, as amended, for the year beginning July 1, 1928, have been distributed. If one desires to continue present narcotic registration, these forms, properly executed, with the proper amount of tax or taxes, must be returned to the office of the Collector of Internal Revenue, 22 Pearl Street, Boston, so that they will reach there on or before July 1, 1928, or penalty will attach.

Persons registering in class 1, 2, 3, or 4 are required to register in class 5 also if they wish to dispense or handle exempt narcotic preparations. No additional payment of tax is necessary, but care should be taken to see that the class 5 block on the application form is checked.

The inventory on Form 713 of narcotics on hand must be prepared under oath or affirmation, in duplicate, the *original* of which is to be kept on file by the maker, and the *duplicate* forwarded to the Collector. No inventory on this form is required for class 1 and 2. An inventory must be made for class 5 if registry is desired in that class. The inventory for class 5 refers only to taxable narcotic drugs which might be set aside for use for manufacturing exempt preparations, and does not require the listing of ready-made preparations and remedies classed as exempt narcotic preparations. In most instances no taxable narcotic drugs are on hand in class 5. If such is the

case, the inventory used for registration in one of the higher classes may also be used for class 5, provided a notation—"No taxable drugs in class 5"—is placed conspicuously on both the original and duplicate copies of the inventory.

Please read the forms carefully before executing them, so that reregistration may be properly accomplished, and to avoid payment of penalty, return all forms with proper remittance in the form of certified check, postal money order, or cash, not later than July 1, 1928.

If there has been a change in address from that at which registration was made last year, please make notation on application, and show date of removal.

In the event that reregistration is not contemplated for any reason, the office of the Collector of Internal Revenue should be so advised prior to July 1.

UNITED STATES CIVIL SERVICE COMMISSION WASHINGTON, D. C.

May 31, 1928.

Subject: Need for Internes in Veterans' Bureau Hospitals.

There is a shortage of eligibles for positions of junior medical officers (internes) in hospitals of the Veterans' Bureau throughout the country. Applications for the positions will be received by the Commission until June 30.

Applicants will not be required to report for examination at any place, but will be rated on their education, training, and experience. Senior students will be admitted to the examination under the conditions specified in the announcement.

Service as interne in a Veterans' Bureau Hospital offers valuable experience to the recent graduate in medicine. The entrance salaries are named in the announcement. At the end of eighteen months' service a salary of $3,300 a year may be paid. Higher-salaried positions are filled through promotion.

Apply to the Commission for detailed information.

Very respectfully,

JOHN T. DOYLE, *Secretary.*

REPORTS AND NOTICES OF MEETINGS

THE ANNUAL MEETING OF THE NEW ENGLAND ROENTGEN RAY SOCIETY

At the annual meeting of the New England Roentgen Ray Society held in Worcester on June 6, 1928, Dr. F. B. Lamb of Portland, Maine, was elected President, Dr. A. S. MacMillan of Boston, Vice-President, Dr. John D. Camp, Secretary-Treasurer, and Dr. M. C. Sosman of Boston, a member of the Executive Committee.

The Secretary reported that eight meetings had been held during the year, three at the Boston Medical Library, and one each at the University Club, the Massachusetts Eye and Ear Hospital, Children's Hospital, Peter Bent Brigham Hospital, and one in Worcester, Mass. Seven new members were accepted during the year, and one member died, Dr. A. P. Merrill of Pittsfield, Mass. There are one hundred and twenty members at present in the Society.

M. C. SOSMAN, M.D.

UNION HOSPITAL IN FALL RIVER

CLINICAL STAFF MEETING

The Regular Monthly Clinical Staff Meeting will be held at the Stevens Clinic on Thursday, June 21, 1928, at 8:15 P. M. All physicians interested are cordially invited. Refreshments will be served.

M. N. TENNIS, M.D., *Secretary to Staff.*

THE SOUTH END NEIGHBORHOOD MEDICAL CLUB

The next meeting of the South End Neighborhood Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, June 19, 1928, at 12 noon.

Dr. Edwin H. Place, Assistant Professor, Tufts Medical School, Professor of Harvard Medical School, and resident physician, City Hospital, will speak on "Diagnosis of Contagious Diseases". All physicians are cordially invited.

The usual luncheon will follow.

SOCIETY MEETINGS

June 16—Annual Meeting of the Massachusetts Nurses' Association. Complete notice appears on page 875.

June 18-20—Meeting of the American Association for the Study of Goiter. See page 875 for complete notice.

June 18-22—Convention of the Catholic Hospital Association. Complete notice appears on page 1597, issue of February 16.

June 19—South End Neighborhood Medical Club. Complete notice elsewhere on this page.

June 21—Union Hospital in Fall River. Detailed notice appears above.

December 3-7—Radiological Society Convention. Detailed notice appears on page 712, issue of May 17.

BOOK REVIEW

Statistical Methods for Research Workers. By R. A. FISHER, SC.D., Formerly Fellow of Gonville and Caius College, Cambridge; Chief Statistician, Rothamstead Experimental Station. Oliver and Boyd. Edinburgh: Tweeddale Court; London: 33 Paternoster Row, E. C. 1928.

The author states that the prime object of this book of 270 pages is to put into the hands of research workers, and especially of biologists, the means of applying statistical tests accurately to numerical data accumulated in their own laboratories or available in the literature.

The book is liberally supplied with the tables which may be useful in making statistical conclusions.

In addition to the consideration of the general purpose of the book, chapters are specifically devoted to the following: Tests of Goodness of Fit, Independence and Homogeneity; Test of Significance of Means, Differences of Means, and Regression Coefficients; The Correlation Co-efficient; Intraclass Correlations and the Analysis of Variance; The Principles of Statistical Estimation.

The author is a master of the latest applications of methods of statistical study. The book has been prepared and edited with great care, but in the presentation of certain matters it would seem that the phraseology might have been better adapted to "persons without special mathematical training", whose needs the book is supposed to meet.